NAVAL POSTGRADUATE SCHOOL
Monterey, California

THESIS

COMPUTER MODELING THE
NEUROPHYSIOLOGY OF VISION

by

Seaborn Montgomery McCrory, III

March 1977

Thesis Advisor: G. Marmont

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE

READ INSTRUCTIONS BEFORE COMPLETING FORM

1. REPORT NUMBER

2. GOVT ACCESSION NO.

3. RECIPIENT'S CATALOG NUMBER

4. TITLE (and Subtitle)
Computer Modeling the Neurophysiology of Vision

5. TYPE OF REPORT & PERIOD COVERED
Master's Thesis; March 1977

6. PERFORMING ORG. REPORT NUMBER

7. AUTHOR(s)
Seaborn Montgomery McCrory, III

8. CONTRACT OR GRANT NUMBER(s)

9. PERFORMING ORGANIZATION NAME AND ADDRESS
Naval Postgraduate School
Monterey, California 93940

10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS

11. CONTROLLING OFFICE NAME AND ADDRESS
Naval Postgraduate School
Monterey, California 93940

12. REPORT DATE
March 1977

13. NUMBER OF PAGES
124

14. MONITORING AGENCY NAME & ADDRESS(if different from Controlling Office)
Naval Postgraduate School
Monterey, California 93940

15. SECURITY CLASS. (of this report)
Unclassified

15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)
Approved for public release; distribution unlimited.

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18. SUPPLEMENTARY NOTES

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)
Visual nervous system
Vision
Neural modeling
Vision modeling
Neurophysiology
Computer modeling
Lateral inhibition

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

The pertinent points of visual neurophysiology and neuroanatomy are reviewed with particular emphasis on how retinal light distributions result in perceived phenomena. The neural modeling techniques used at the Naval Postgraduate School are described. These stem from a basic model capable of calculating the postsynaptic potential and spike outputs for any sequence of excitatory and inhibitory

DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE
S/N 0102-014-6601 |

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

inputs. More advanced programs model facilitation, fatigue, and narrow band motion detection. The most advanced program models lateral inhibition in an eight neuron linear array. The lateral inhibition network is used to show temporal phenomena (null vs. preferred direction, fast vs. slow speed detection) as well as spatial phenomena (Mach bands, line sharpening, disinhibition, spatial frequency response). Many suggestions for future modeling work are given.

Approved for public release; distribution unlimited.

COMPUTER MODELING THE NEUROPHYSIOLOGY OF VISION

by

Seaborn Montgomery McCrory, III
Lieutenant Commander, United States Navy
B.S., United States Naval Academy, 1965

Submitted in partial fulfillment of the
requirements for the degree of

MASTER OF SCIENCE IN ELECTRICAL ENGINEERING

from the

NAVAL POSTGRADUATE SCHOOL

March 1977

ABSTRACT

The pertinent points of visual neurophysiology and neuroanatcmy are reviewed with particular emphasis on how retinal light distributions result in perceived phenomena. The neural modeling techniques used at the Naval Postgraduate School are discussed. The specific computer programs used by the author in modeling are described. These stem from a basic model capable of calculating the postsynaptic potential and spike outputs for any sequence of excitatory and inhibitory inputs. More advanced programs model facilitatien, fatigue, and narrow band motion detection. The most advanced program models lateral inhibitien in an eight neuren linear array. The lateral inhibition network is used to show temporal phenomena (null vs. preferred direction, fast vs. slow speed detection) as well as spatial phenomena (Mach bands, line sharpening, disinhibition, spatial frequency response). Many suggestions for future modeling work are given.

TABLE OF CONTENTS

I. INTRODUCTION..... 11

II. BACKGROUND..... 13

A. THE NEURON AND THE SYNAPSE..... 13

B. THE VISUAL PATHWAYS..... 15

C. THE TARGET NEURON CONCEPT..... 16

1. Retinal..... 19

2. Geniculate..... 19

3. Cortical..... 19

4. Pontine..... 20

D. THE RETINA..... 21

E. SPATIAL CONSIDERATIONS..... 25

1. Sharpening..... 25

2. Spatial Frequency..... 25

3. The Spatial Modulation Transfer Function..... 26

III. THE MODELS..... 28

A. THE COMPUTER FACILITY..... 28

B. THE BASIC NEURON MODEL..... 29

C. THE TARGET NEURON..... 31

D. THE NEURAL OSCILLATOR..... 35

E. ADAPTIVE NEURON..... 36

F. LATERAL INHIBITION..... 39

G. INLIB AND OUTLIB..... 43

IV. RESULTS..... 47

A. CONVENTIONS FOR PLOTTING OF DATA..... 47

B. TEMPORAL PHENOMENA..... 48

1. Fatigue and Facilitation..... 48

2. The Neural Oscillator..... 53

3. The Target Neuron..... 53

4. Motion Detection..... 54

a. Band Pass..... 60
b. Null versus Preferred Direction. 64
c. Fast Pass..... 67
d. Slow Pass..... 75
5. Regularization of Random Inputs..... 75
C. SPATIAL PHENOMENA..... 82
1. Basic Spatial Behavior of LINHIB.... 82
2. Inhibition and Disinhibition..... 82
3. Bright Line Phenomena..... 87
a. Line Sharpening by Inhibition... 87
b. Extreme Phase Sensitivity..... 91
c. Line Shift..... 91
d. Optimum Stimulus Representation. 92
e. Line Sharpening by Threshold.... 92
4. Spatial Frequency Response..... 102
a. Suppression of Low Spatial Frequencies..... 103
b. Mach Bands..... 108
V. CONCLUSIONS..... 112
VI. RECOMMENDATIONS FOR FUTURE MODELING WORK.... 115
LIST OF REFERENCES..... 121
INITIAL DISTRIBUTION LIST..... 123
LIST OF FIGURES..... 7

LIST OF FIGURES

1. Neuron and Synapse..... 17
2. Visual Pathways..... 18
3. The Retina..... 24
4. Basic Neuron and Neural Circuit..... 32
5. Flow Chart of EIPSP..... 33
6. Stimuli with Decreasing Potency vs. Time..... 34
7. LINHIB Network, Constant Delay..... 44
8. LINHIB Network, Ramp Delay..... 45
9. Flow Chart of LINHIB..... 46
10. Fatigue of E-Inputs..... 50
11. Fatigue of I-Inputs..... 51
12. Facilitation..... 52
13. Sinusoidal PSP, Spontaneous..... 55
14. Sinusoidal PSP, Random Inputs..... 56
15. Instability..... 57
16. High Frequency Sinusoidal PSP..... 58
17. Regularization by Sinusoidal PSP..... 59
18. LINHIB with Downward Target, Part 1..... 61
19. LINHIB with Downward Target, Part 2..... 62
20. Speed Band Pass Detector..... 63

21. Null versus Preferred Direction, Part 1..... 68

22. Null versus Preferred Direction, Part 2..... 69

23. Null versus Preferred Direction, Part 3..... 70

24. Circuit for Null vs. Preferred Direction..... 71

25. Fast Pass Detector, Part 1..... 72

26. Fast Pass Detector, Part 2..... 73

27. Fast Pass Detector, Part 3..... 74

28. Slow Pass Detector, Slow Target..... 77

29. Slow Pass Detector, Bright Fast Target..... 78

30. Slow Pass Detector, Normal Fast Target..... 79

31. Regularization by LINHIB, Part 1..... 80

32. Regularization by LINHIB, Part 2..... 81

33. Basic Spatial Behavior of LINHIB, Part 1..... 83

34. Basic Spatial Behavior of LINHIB, Part 2..... 84

35. Inhibition and Disinhibition, Part 1..... 85

36. Inhibition and Disinhibition, Part 2..... 86

37. Line Sharpening, Regular Inputs, Part 1..... 93

38. Line Sharpening, Regular Inputs, Part 2..... 94

39. Line Sharpening, Regular Inputs, Part 3..... 95

40. Line Sharpening, Random, Part 1..... 96

41. Line Sharpening, Random, Part 2..... 97

42. Line Sharpening, Random, Part 3..... 98

43. Abnormal Phase Sensitivity, Part 1..... 99

44. Abnormal Phase Sensitivity, Part 2.....100

45. Line Shift.....101

46. Low Spatial Frequency Response, Dim.....104

47. Low Spatial Frequency Response, Bright.....105

48. Mach Bands.....110

49. Absence of Mach Bands.....111

ACKNOWLEDGEMENT

I wish to thank Professor George Marmont for providing the education in neurophysiology, the wonderful computing facility, the atmosphere of relaxed diligence, the proper attitude toward claimed but unproven new discoveries, and his continuing interest in his students. Without him, neither this thesis nor the work by other students which preceeded it would have been possible.

I wish also to thank LT. Dennis Marvel for the writing of the original version of the lateral inhibition program, LT. Dan Lashbrook for his assistance in understanding and modifying the program, and LT. Billy Cornett and LCDR. Jean Fricke for their independent work on neural reverberating circuit models.

Without my wife, Pamela, and daughters Amanda and Courtney, none of this would have as much meaning.

I. INTRODUCTION

The visual system is a truly amazing thing. It must, as is true of anything having to do with living organisms, be fully describable both anatomically and functionally in terms of well defined physical principles. This is certainly true at present of the optical end of the visual system, from the cornea to the retina. There is very little mystery to the refraction , reflection, and diffraction of light in the structures of the eyeball. But there is still a great deal of mystery associated with the nervous system end of the visual system, even though much hard, reproducible scientific data exists.

These data tend to be of four general types. In animals, data consists largely of microelectrode studies of cellular electrical activity in response to various visual or electrical stimuli. In humans it is generally perceptual in nature , the subject being presented with various visual stimuli at or near the threshold of detection and being asked for a verbal response upon detecting the stimulus. One exception is the electroencephalogram, recorded using scalp electrodes as the subject is presented with various stimuli. Of course, there are the all-important histological studies of both human and animal nervous tissue, whose aim, in general, is to ascertain functional anatomical features, such as interconnections between neurons. All four of these data types are of great value in aiming toward a solution of how visual systems work. But what has been conspicuously missing until recently are suggestions and speculations as to what circuits of neural elements could be responsible for visual phenomena. It is the aim of this thesis to review

what is known about the neurophysiology of visual systems, and then to propose some models of neurons and neural circuits which could explain some of the observed perceptual phenomena. It must be restated for emphasis that the results of the modeling in this thesis, although based upon the best information available, are necessarily highly speculative, and should be considered as how certain phenomena *might* occur rather than how they *do* occur.

II. BACKGROUND

The process we call vision begins when photons from a scene enter the cornea and ends when the visual association area of the brain conveys meaning to the organism. It is obvious that a vast amount of information processing occurs in order for this transition to occur. Much information which enters the cornea is lost to the organism, but this is an adaptive advantage rather than a disadvantage. The organism must respond quickly to the important features of the visual field. If all incident information were required to be processed and presented to the nervous system of the organism, the response could not be as rapid. It is apparent, therefore, that visual systems must have means for extracting the important aspects of a scene. The aspects which are considered important vary from species to species, but two things tend to be extracted by the visual systems of all organisms studied: high contrast features and movement.

A. THE NEURON AND THE SYNAPSE

Before discussing the processing of information by the visual system, the basic functional units of which all nervous tissue is built will be described.

The neuron, or nerve cell, consists of a cell body (soma), an input end (dendrites), and an output end (axon), as shown in Fig 1. The point at which two nerve cells communicate is called a synapse. Consider a nerve impulse (action potential) proceeding to the right at point A. Upon

reaching the dendrites of neuron 1, the propagating action potential causes some submicroscopic change which causes the neurotransmitter vessicles to dump their contents into the synaptic cleft. Mitochondria are present in the bouton to provide the energy necessary for this release. After diffusing across the cleft, the neurotransmitter causes ion permeability changes in the subsynaptic membrane which in turn cause membrane potential changes.

The membrane would have a resting potential of -80 millivolts (inside negative with respect to the outside). The effect of an excitatory neurotransmitter would be to depolarize the membrane.

By a process called electrotonic spread, the potential change at the dendrites is transmitted by ion currents to the axon hillock. If the membrane potential at the axon hillock reaches roughly -60 millivolts, a dramatic change in membrane permeability to ions causes a propagating polarity reversal (the action potential), which travels the length of the axon of neuron four.

If other E-inputs are occurring, say at neuron two, spatial summation would occur. This means that inputs at different dendritic locations will each cause a change at the axon hillock, and can work together, or sum, to produce an action potential.

If a time sequence of E-inputs occurs at one dendrite, temporal summation occurs. This means that inputs at different times can work together, or sum, to produce an action potential, so long as the inputs are not too far apart in time. Figure 1 shows a typical post synaptic response (PSR), which is the change in membrane potential at the axon hillock due to a single input at a dendrite. The finite duration of the PSR means that inputs occurring in

rapid succession will not each have to start anew from rest potential, but will have some potential change left over from previous inputs.

Inhibitory as well as excitatory neurons exist. If a neuron is inhibitory, it releases a neurotransmitter which causes postsynaptic hyperpolarization vice depolarization. That is, it tends to inhibit downstream neurons from producing outputs. The shape of the inhibitory PSR is an inverted version of the excitatory PSR, and summation at the axon hillock is a subtraction. A neuron is either wholly excitatory or wholly inhibitory--never mixed.

B. THE VISUAL PATHWAYS

Figure 2 depicts the visual processing pathways. Light enters the eyes and is focused on the retina by the cornea and lens structures. As quanta of light are absorbed, the retinal receptors respond by varying membrane potentials. These varying potentials are coupled through the cells of the retina to the ganglion cells, whose fibers form the optic nerve. The ganglion cells code the visual scene into spike action potentials for transmission to the brain. The information leaving the retina has been processed by the retinal neural circuits, and therefore is not simply a coded version of the scene.

At the optic chiasm, fibers undergo a rerouting such that all information coming from the right half of the visual scene goes to the left half of the brain, while information coming from the left half of the visual scene goes to the right half of the brain.

At the lateral geniculate nuclei, the fibers synapse

with other neurons. The neural interconnections in the lateral geniculate nuclei process the visual scene even more. It is believed that the interleaving of information in the lateral geniculate nuclei from the same scene portion but from different eyes constitutes processing which ultimately results in depth perception.

Fibers from the neurons of the lateral geniculate nuclei terminate on neurons of the visual cortex. Here, the processing has proceeded to the degree that specific visual features at specific locations are being extracted. These specific features of the visual scene are pieced together in the visual association areas, and ultimately result in the organism's being able to interact with his visual environment.

Not shown is the pons of the brain, located in the brain stem. The pons also receives visual information and is an important center for processing this information.

C. THE TARGET NEURON CONCEPT

The presence of a particular feature is nearly always signalled by strong spike outputs from a single neuron. This neuron is usually retinal (a ganglion cell), geniculate, cortical, or pontine, and is termed a target neuron in this thesis, since much information is targeted at or converged to the neuron. The output tends to be binary in nature--either the condition is met, in which case frequent outputs occur, or the condition is not met, in which case few or no outputs occur. In general, the simplest sets of criteria for output production occur early in the visual pathway, while very complicated sets of criteria generally apply later.

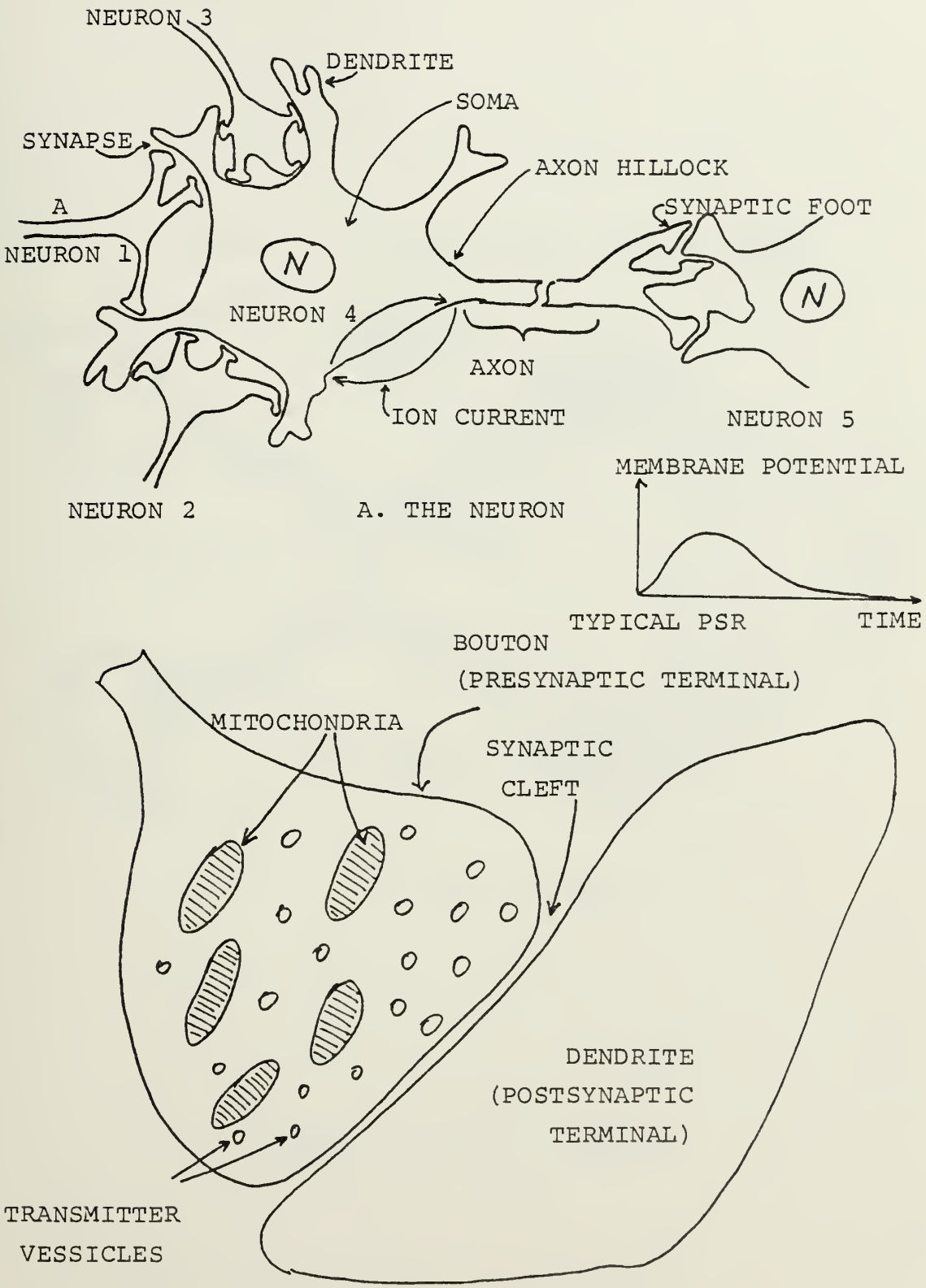


FIGURE 1 - NEURON AND SYNAPSE

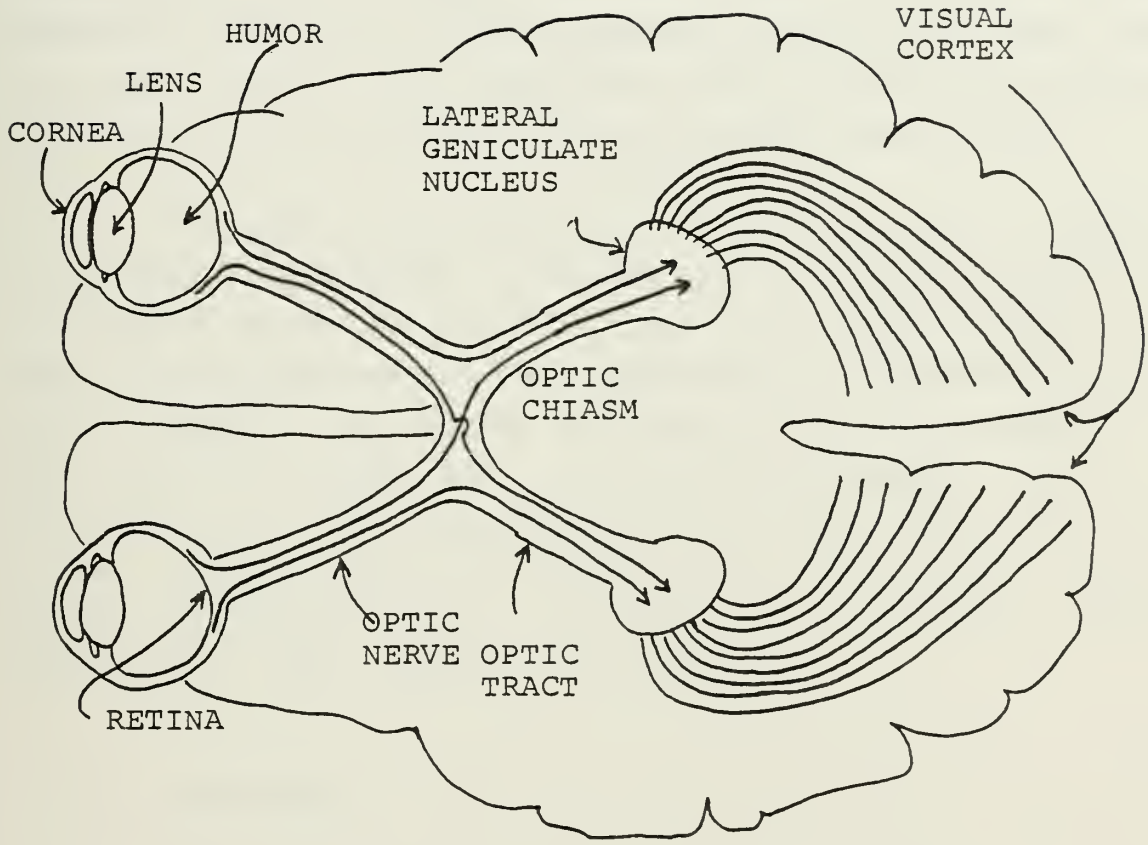


FIGURE 2 - VISUAL PATHWAYS

1. Retinal

An example of a simple criterion is common for ganglion and geniculate cells. Termed the "on-center" cells, strong outputs occur when receptors feeding them are illuminated by a small dot of light. The dual of this situation occurs for the "off-center" cells--the small dot of light causes the ganglion cell to have no outputs, whereas in tctal darkness, the cell would have outputs.

In addition to simply turning on or off in response to a small dct of light, ganglion cells respond oppositely to bright surround stimuli. That is, when an annulus of light illuminates receptors surrounding the receptors which feed a ganglion cell of the on-center type, the result is an off response. Similarly, a surround stimulus for an off-center ganglion cell produces an on response. The cause of the surround stimulus effects has been shown to be inhibitory interneurons (horizontal and amacrine cells) which spread their inhibition laterally.

2. Geniculate

Geniculate cells remain fairly simple in their properties. They are very similar to ganglion cells, but have an enhanced ability for surround stimuli to counteract center stimuli. Thus they appear to represent a higher degree of capability for extracting information concerning contrast.

3. Cortical

Examples of more involved sets of criteria occur cortically, where "simple" as well as "complex" cells have been established [Ref 10]. Simple cells respond most favorably to line stimuli which have the correct position in the visual field and the correct angular orientation. Complex cells respond strongly for line stimuli with proper orientation and direction of movement, and tend to have larger "on" fields. Complex cells are believed to be targets for outputs of many simple cells which detect the same orientation. In some way (possibly unilateral spread of inhibition) the neural interconnections mediate the motion direction specificity of the complex cells. Thus the simple and complex cortical cells present signals to the association areas, each signal having its own meaning in terms of the content of the visual field. Signals from many millions of these cells interconnect with many millions of association cells to provide the organism with a conscious perception of the scene.

4. Pontine

The pons of the brain is also an important target area for visual information. It is believed that the pons is important in assembling visual information from the visual cortex and relaying this information to the cerebellum for use in motor control [Ref 8]. A typical pontine cell responds strongly to motion in one direction only and is only weakly stimulated or even inhibited by motion in the opposite direction. Small stimuli are most effective in producing strong outputs; shape and orientation are not generally important. An individual target cell would respond most strongly to motion at one particular angular velocity. The total range of angular velocities covered by all cells recorded was roughly ten to 800 degrees per second [Ref 8].

D. THE RETINA

Because the response of retinal ganglion cells is not a simple function of photons falling on connected receptors, it is apparent that retinal processing does indeed go on. The retina is important enough to deserve a description here, however brief and oversimplified, because it contains two systems of laterally inhibiting interneurons. Lateral inhibition is very fundamental to the functioning nervous system.

The retina is pictured in Fig 3 in cross section. Light illuminates the receptor cells after passing through the eyeball and the other neural layers. Photon absorptions cause changes in visual pigment substances which in turn cause variations in the membrane potential of the receptor cells. Recent evidence indicates that this membrane potential variation is coupled to the bipolar and horizontal cells by modulating the flow of an unidentified chemical neurotransmitter substance. The bipolar and horizontal cells are very small in length as compared with normal "long-axon" neurons, and therefore do not need to employ spikes to transmit information along their length. The information is in the form of subthreshold membrane potential changes (slow potentials) which travel electrotonically and modulate the release of a neurotransmitter just as did receptor potential variations. Bipolar cells terminate on amacrine and ganglion cells (in primates). Amacrine cells are larger, depolarize when stimulated (receptors, horizontal, and bipolar cells hyperpolarize), and tend to have a propensity for spike production. Horizontal and amacrine cells are inhibitory, while the other retinal cells are excitatory. The axons of ganglion cells form the optic nerve, in which all

information is in the form of action potentials. Because no other connection exists between the retina and the brain, these action potentials constitute the total result of retinal processing of the visual scene.

Several important points need to be made about the neural interconnections just discussed. Each ganglion cell has associated with it a receptive field. Foveally, there are few receptors feeding each ganglion cell. These receptors are exclusively cones, and fairly high levels of light are required for vision. Peripherally, as many as a hundred receptors, mostly rods, feed each ganglion cell, thus enhancing sensitivity at the expense of acuity. The most important point, however, is that the information leaving a certain ganglion cell depends not merely upon the brightness falling upon the receptors feeding it, but also depends upon what light distribution falls upon adjacent areas. The horizontal and amacrine cells perform this function by extending laterally, and by inhibiting the neurons upon which they terminate. This explains the term "lateral inhibition," and also explains why ganglion cells respond oppositely to bright surround stimuli.

Another important type of processing which occurs retinally is the development of combined color signals. Each of three types of cone cells has its own response spectrum, with significant overlap. Ultimately, the signals from the three receptor types must be compared to give the organism the proper color sensation. There is evidence that this comparison begins retinally by the formation of difference signals at the ganglion cell level. Although color visual processing will not be modeled in this thesis, this was mentioned to point out that other types of processing do go on retinally. Additionally, this is another example of how visual processing occurs in stages, with early development occurring retinally, and final features being extracted

cortically.

Thus the retina is capable of coding the visual scene into action potentials which go to the brain. The coding is the result of the total spatial distribution of light on the retina--not merely on each local point of light taken independently. This lays the ground work for modeling of spatial phenomena later in this thesis.

The response of a ganglion cell can reflect temporal properties as well as spatial distributions of light. In the mudpuppy (a small fish), microelectrode measurements have confirmed that one type of ganglion cell responds to stationary light distributions while a different type of ganglion cell responds only to changes in light distributions. This means that retinal processing has occurred which has extracted motion information from the visual field. In the frog, there are five types of ganglion cells, one of which is very specific for a small, convex, dark object (such as a fly) moving through the receptive field of that cell. This represents a very high degree of motion detection capability located right in the frog's retina. In other vertebrates, similar target cells exist, but at higher centers such as the pons, the superior colliculus, and the visual cortex.

The point of discussing retinal motion detectors is to point out that since the "equipment" and types of neural interconnections available retinally are very well known, the number of ways in which this system can work are limited. A lateral inhibition network can be used to model a motion detector. Furthermore, the presence of such a retinal motion detecting system in the frog lends credence to any such model.

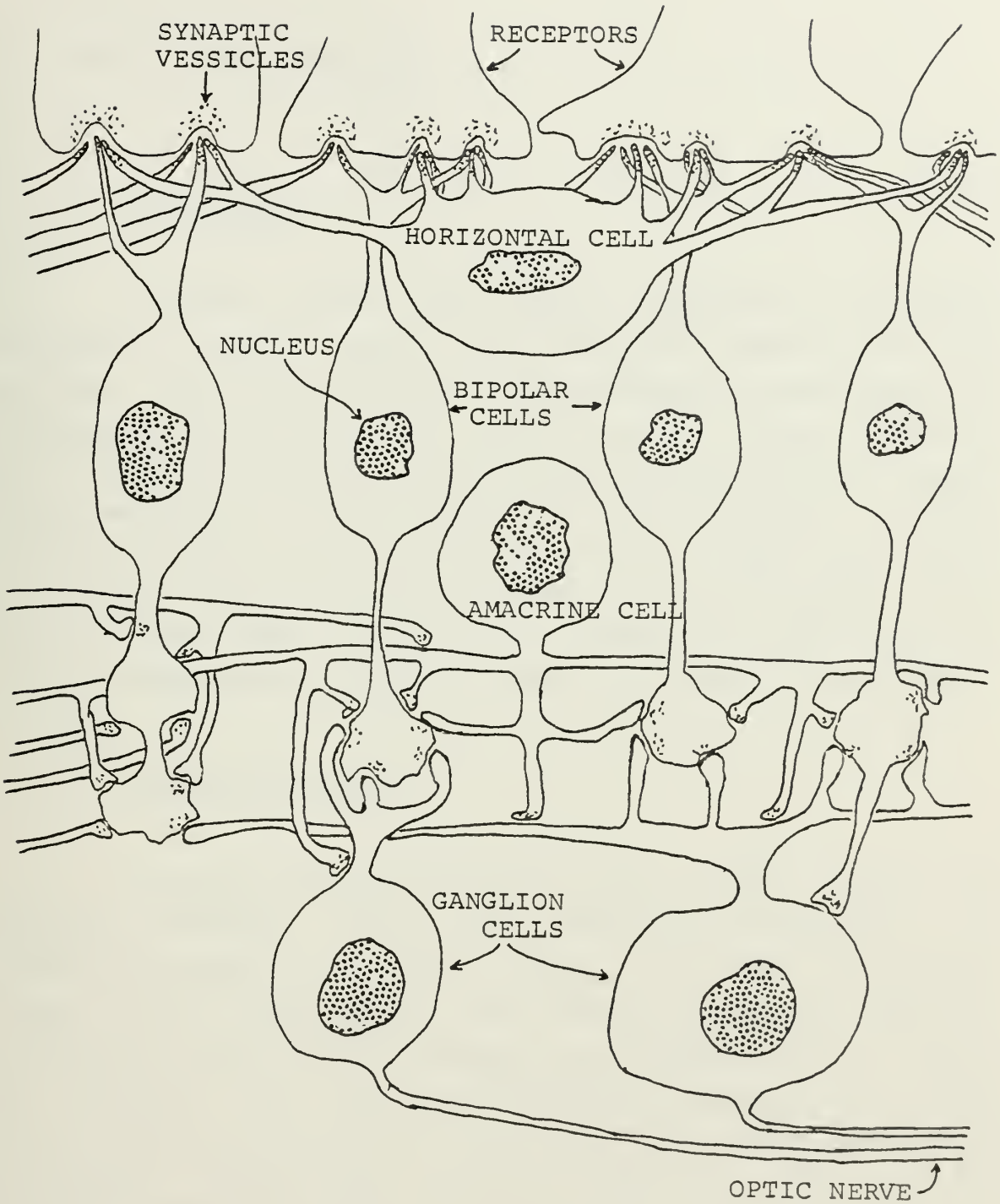


FIGURE 3 - THE RETINA

E. SPATIAL CONSIDERATIONS

1. Sharpening

A neural network with lateral inhibition can sharpen details in a visual scene. The entry of light into the optical end of the eyeball is accompanied by scattering and diffraction, both of which tend to create a blurred image on the retina of an object which should be perceived as having sharp contrasts. The end result of this process is that cortical cells responsible for detecting a sharp line, edge, or slit at a certain angular orientation at a certain point in the visual field can do so more efficiently. This process will be explained more fully later, as models are presented.

2. Spatial Frequency

A useful concept in dealing with visual scenes is spatial frequency. This term refers to the frequency, in cycles per degree of visual angle, at which luminosity varies from light to dark. The simplest case is a level of luminosity which does not change with position (zero cycles per degree). The next most fundamental example is a field where there is no variation vertically, but horizontally the luminosity varies from light to dark as the sine of position. This is called a sinusoidal grating. Another example is a series of vertical light and dark bars, the so-called square wave grating. Just as with any periodic time series waveform, these periodic spatial gratings can be considered as being the sum of all Fourier components, each component being a pure sine at some harmonic of the

fundamental frequency.

3. The Spatial Modulation Transfer Function

The visual system responds differently to different spatial frequencies. In perceptual studies, the subject is asked to signal when he just detects a sinusoidal grating flashed on a screen. Each grating flashed has two basic properties: spatial frequency and contrast. Contrast refers to the difference in luminosity between the lightest and darkest regions. When the data is plotted, it is found that roughly three cycles per degree is the spatial frequency at which subjects can detect gratings with the lowest contrast. As spatial frequency gets higher or lower than three cycles per degree, more contrast is required for detection. The resulting curve of spatial frequency versus contrast threshold for detection is called the spatial modulation transfer function. It is considered as the best estimate of the spatial frequency response of the human visual system.

The high frequency fall-off of the spatial modulation transfer function is due in part to the optical properties of the cornea, lens, and humor of the eye. Any lens system has an upper limit to its resolution, and transfer functions have long been used to describe the high spatial frequency performance of lens systems. The finite size of the individual receptors, the scattering of light as it impinges upon the retina, and the summation from receptors to bipolar cells are all believed to play roles in limiting the high frequency response.

The low frequency fall-off is much harder to comprehend, and involves a comparison of the visual angle subtended by one cycle of pattern with the visual angle through which inhibition from a pinpoint of light would

spread. When inhibition spreads through one-half cycle, outputs from ganglion cells are at enhanced contrast levels. This is because strong inhibition from the bright area inhibits the dark area, while lack of inhibition from the dark area allows maximum output from the bright area, thus enhancing contrast. It is important to note here that since inhibition does not begin until several neurons are traversed radially, the centers of the bright peaks do not inhibit the edges of the bright regions. Next, consider a situation in which the lateral extent of inhibitory spread is much less than one cycle of pattern. The result here would be that bright areas inhibit themselves while dark areas receive little inhibition from themselves. The effect would be a loss of contrast.

The low response at low frequencies as well as the contrast enhancing response at high frequencies will be modeled in this thesis.

III. THE MODELS

A. THE COMPUTER FACILITY

The computer modeling for this thesis was all performed using the PDP-11/40 digital computer, made by Digital Equipment Corporation of Maynard, Mass., and located in the Bioengineering Laboratory at the Naval Postgraduate School. This computer is equipped with 32K words (1K=1024, one word equals 16 bits) of readily addressable core memory, two RK-11 disk drives, a TC-11 magnetic tape drive pair, and an LA-30 keyboard/character printer. Block data is displayed on a storage cathode ray tube and plotted by a Hewlett Packard 7004B X-Y recorder.

All neural modeling programs in existence when inherited by the author were written in Time Series Language (TSL) with links to machine language routines where necessary. TSL is a higher level language especially designed for manipulating blocks of data. There are advantages as well as disadvantages to using a higher level language in general and TSL specifically. On the positive side, programs are easy to change, as moving of statements is done automatically. Also, the programmer need know nothing about the processor and memory utilization in order to perform certain limited programs. But in order to extract the fullest performance from the 32K words of core, speed program execution somewhat, know exactly where in core stacks and blocks are located, and generally know what is happening, there is no substitute for complete machine

language programming. To this end, Dr. Marmont has painstakingly developed the APTEC "language," which is a collection of frequently used data processing and servicing routines. All neural modeling programs used in this thesis use APTEC and pure machine language.

B. THE BASIC NEURON MODEL

The object of the basic excitatory and inhibitory postsynaptic potential (EIPSP) program is to form the proper PSP response given a scenario of excitatory and inhibitory inputs at a neuron's dendrites (Fig 4a). The accomplishment of this rather modest sounding task took considerable time, talent, and guidance, and was accomplished by the students of Dr. Marmont during 1975 as work for his course in computer modeling. The operation of the EIPSP program will be reviewed here, since it forms the basis upon which later more elaborate modeling programs were built.

First, the properly shaped responses for excitatory as well as inhibitory inputs are formed in a pair of 1K blocks. These are termed the excitatory and inhibitory postsynaptic responses (EPSR and IPSR) and are shown in Fig 10. Then a 1K PSP processing block is established along with a pointing register for use in forming the PSP at each word of the block. Also needed are excitatory and inhibitory input counting registers, and two stacks for keeping track of the proper age of each input which has occurred since the last output.

Processing begins by testing for the presence of an excitatory input at the first word of the block (Fig 5). If an input is present, a zero age marker is placed on the E-age stack, and the E-counter is incremented. The E-counter

is then tested to determine whether any inputs requiring PSP update have occurred since the last output. If the counter is positive a nifty maneuver forming the heart of EIPSP is accomplished. Using the E-input counter as a loop counter and the E-age stack as the source of ages for each E-input which has occurred since the last output, the PSP word is formed by adding together the proper number of properly aged EPSR's. The E-age stack is then aged by one word to prepare it for the next processing pass.

This identical procedure is followed for I-inputs, except that the aged IPSR's are subtracted instead of added when forming the PSP word.

Next the resultant PSP word is compared with the threshold for spike initiation. If below threshold, processing proceeds to the next word of the block. If equal to or greater than threshold, a marker is placed in the output block, the E and I-age stacks are reset, the E and I-input counters are zeroed, and the processing block pointer is advanced to the end of the refractory period. A test is then made to determine whether or not the pointer is at the end of the processing block. If so, processing ends and the results are displayed. If not, the pointer is advanced to the next word of the block and the entire sequence repeated.

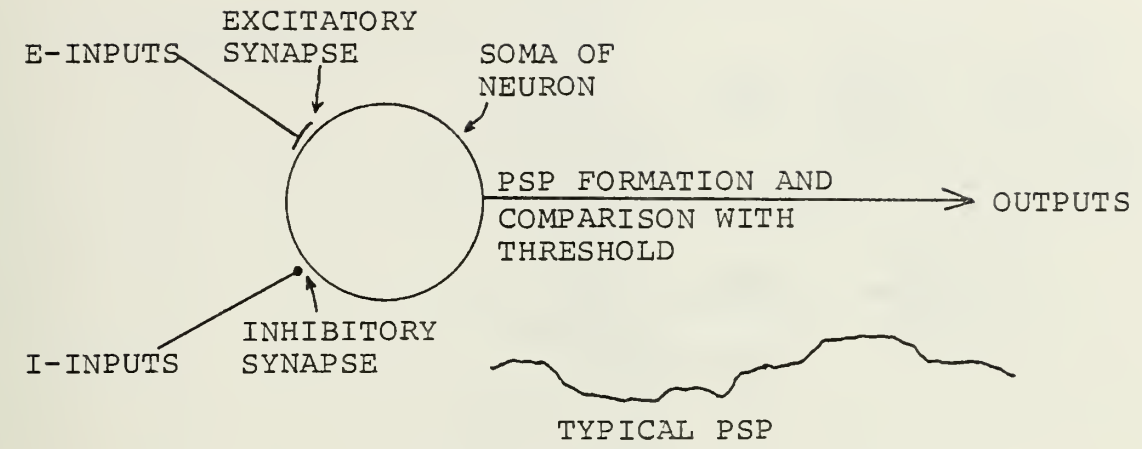
No effort was made to model the membrane action potential which exists during output. Rather, no processing whatever is done during this time, leaving the PSP equal to zero. (The PSP processing block was set to zero prior to beginning processing). The action potential, if modeled, would be strictly cosmetic, since no use would be made of the membrane potential excursion which would occur.

This basic neural model is extremely useful in building

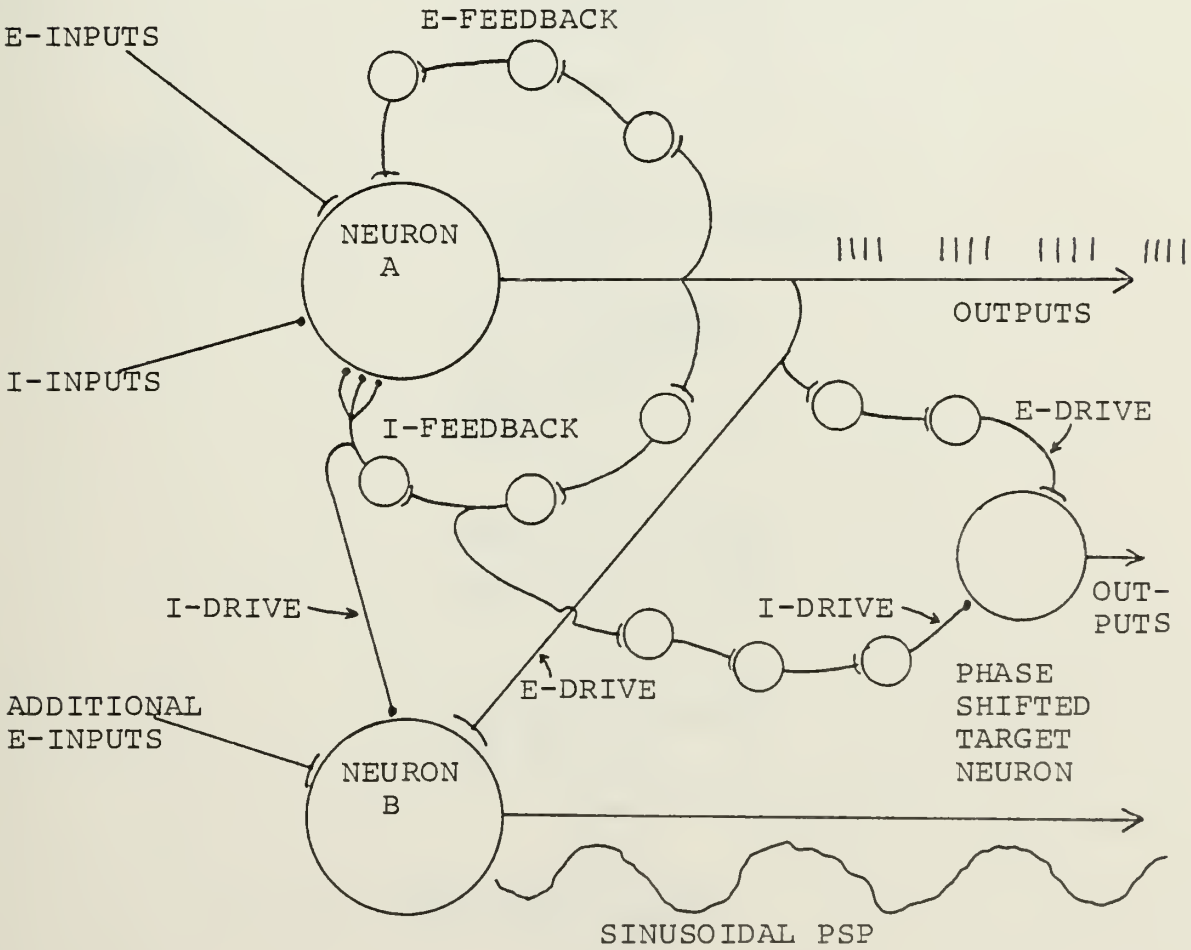
functional neural circuits. This is done in its simplest form by storing the 1K output marker block on disk and employing that as an excitatory or inhibitory input to subsequent neurons, thus modeling divergence. Similarly, more than one output block can be added together and applied as the E or I-input to a subsequent neuron or neurons, thus modeling convergence. In its most complex form, the eight basic neurons are combined into a simultaneous, interactive lateral inhibition network, to be described later.

C. THE TARGET NEURON

NEUROB is the computer program which models the target neuron. Target neurons in visual processing are probably in the retina, lateral geniculate nucleus, pons, or the visual cortex, and perform specific tasks, such as firing for one very specific visual stimulus. Neuron B (Fig 4b) represents a target neuron which fires when its E-inputs coincide with peaks in a sinusoidally oscillating PSP. Such a neuron must have the oscillating PSP created in some way; in neuron B, this is done by E and I-inputs provided by outputs from an oscillator, neuron A. There are reasons for postulating the existence of an element such as neuron B. First, the frequency of oscillation of the PSP could be related in some way to the frequencies preferentially present in the electroencephalogram during some task [Ref 11]. Second, sinusoidally varying PSP's have been observed in nature [Ref 2]. In this thesis, the additional E-inputs come from the summed outputs of the eight channels of the lateral inhibition program; neuron B has copious outputs when these additional E-inputs occur at the right frequency. This condition will correspond to a small target crossing the visual field at the preferred angular velocity.



A. SIMPLE NEURON MODELED BY EIPSP



B. OSCILLATORY NEURON WITH TARGETS

FIGURE 4 - BASIC NEURON AND NEURAL CIRCUIT

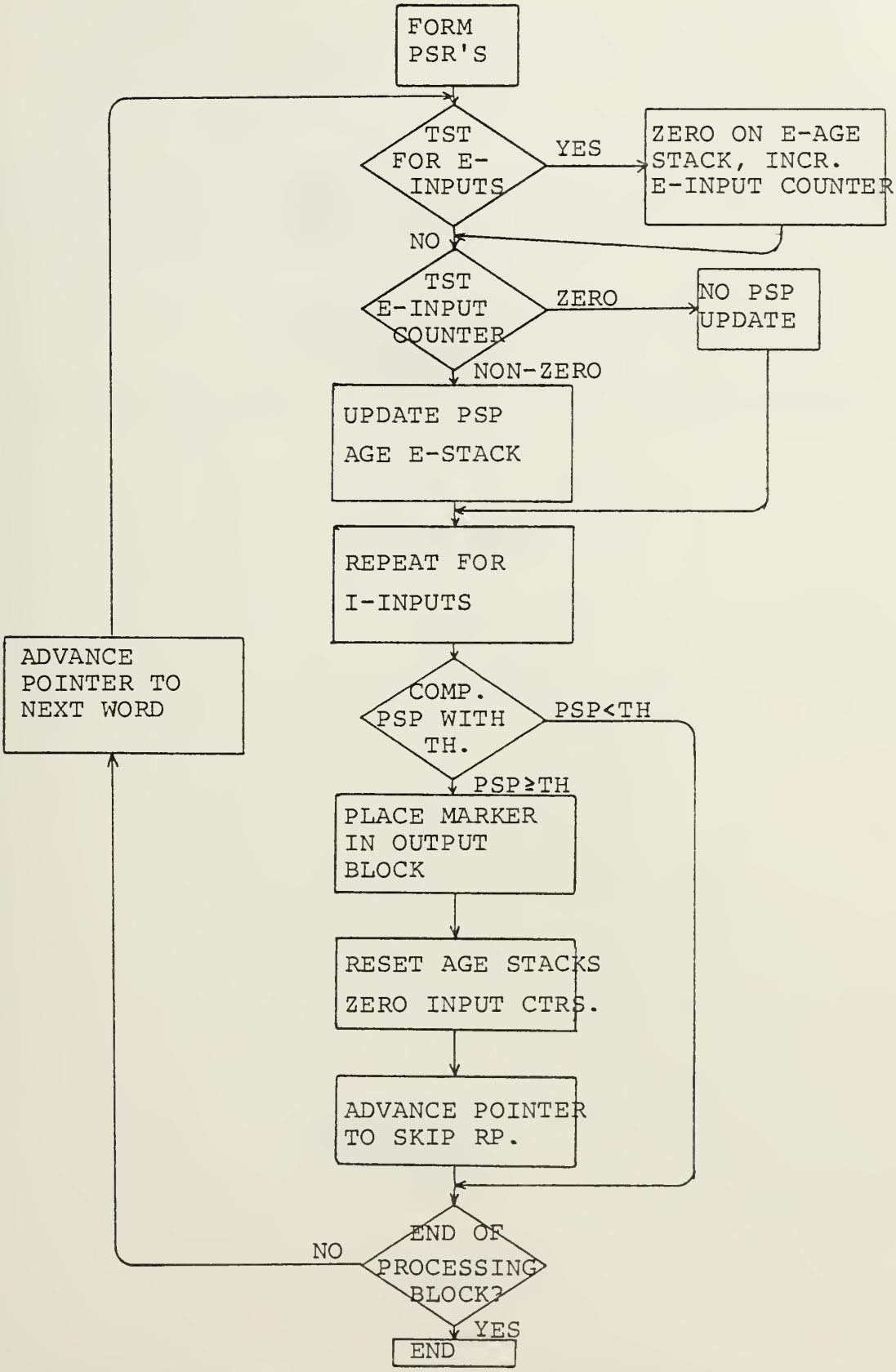
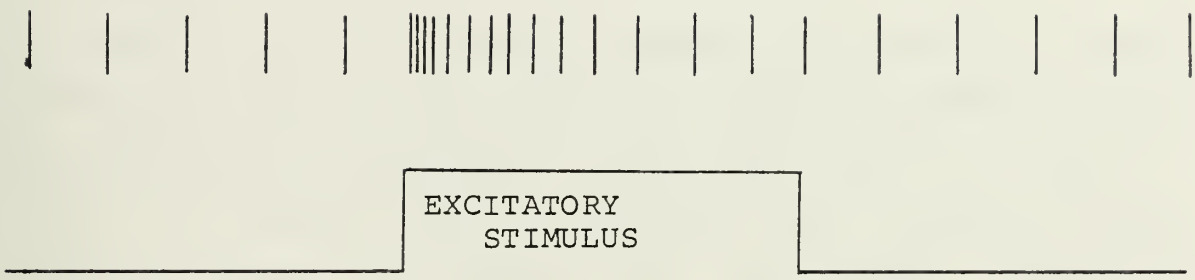
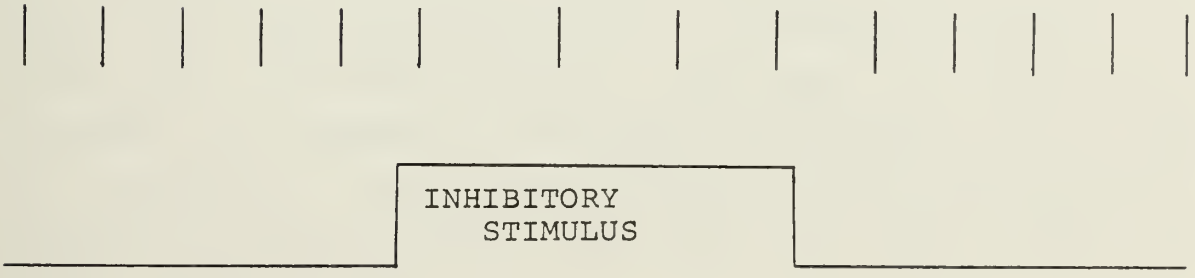


FIGURE 5 - FLOW CHART OF EIPSP



A. EXCITATORY



B. INHIBITORY

FIGURE 6 - STIMULI WITH DECREASING POTENCY VS. TIME

D. THE NEURAL OSCILLATOR

NEUOSC is the computer program which models a neuron (neuron A, Fig 4a) which generates regularly spaced groups of outputs. These outputs are then used to drive the PSP of target neuron B. NEUOSC is versatile in that it allows a choice of several ways of producing these regularly spaced output groupings. All methods are, of course, compatible with how neurons have been observed to function. Neuron A works by first being excited to threshold and producing output. This is done by E-inputs at the dendrites, by self excitability, or by a combination of the two. The outputs are then fed back as inhibitory inputs after a specifyable time delay. By choosing the right excitation level, inhibitory feedback delay, and inhibitory feedback strength, the neuron can be made to provide bunched output pulse trains where the bunches occur at any desired frequency.

Self excitability has been observed in microelectrode studies to be of either sawtooth or sinusoidal form [Ref 2]. All programs in this thesis utilize the sawtooth form, which is generated in the program by forming a ramp in the PSP block. The ramp begins following a refractory period and ends when an output occurs. The slope of the ramp is callable as a parameter. The outputs from neuron A are used as E-inputs to drive neuron B's PSP, while the inhibitory feedback pulses of neuron A are used as the I-inputs to drive neuron B's PSP.

The use of excitatory as well as inhibitory driving allows a much higher frequency in the PSP of neuron B. By using excitatory inputs alone to drive the PSP, the frequency at which a significant amplitude is attainable is

limited by the exponential fading of the postsynaptic response. By alternately driving with I-inputs, the sharp rise of the inhibitory postsynaptic response curve is employed to quickly drive the PSP below rest potential, thus speeding up the oscillation.

In addition to the normal modes of operation, neuron A is capable of accepting external I-inputs, and also of excitatory feedback, which might be of future use in modeling excessive neural discharge, such as might occur during epilepsy.

E. ADAPTIVE NEURON

PSPFAT is a computer program which models the behavior of a neuron whose parameters change with time, or adapts to its inputs.

The underlying purpose for modeling an adaptive neuron was to have a neural circuit element which would fire for only a brief burst following a step input of excitation. This would be roughly equivalent to a neural differentiator, although the behavior when the step input is terminated is inconsistent with this analogy (the neuron does not go to a negative firing rate due to a negative rate of change of inputs). The adaptive neuron would serve as a means to separate change from steady state, and would have strong output only immediately following change. Such a neuron might be useful in a motion detecting network where a bright, moving spot of light were involved.

Neurons have been observed which have a certain low firing rate due to self-excitability under quiescent (no input) conditions [Ref 2]. When provided with an excitatory

stimulus, the firing rate is at first rapid, but then dies off to a level only slightly above the quiescent rate (Fig 6a). When the excitatory stimulus is terminated, firing returns to the quiescent rate. When an inhibitory stimulus is turned on, the firing rate goes to zero initially, but then returns to a rate slightly below the quiescent rate. Termination of the inhibitory stimulus causes the firing rate to return to quiescent.

Before proceeding with PSPFAT, a good deal of thought was put into exactly what physical process is responsible for the phenomenon being modeled. When a neuron is referred to as less excitable, this normally means the membrane is hyperpolarized; being more excitable normally refers to the membrane being depolarized. These conditions are fully modeled by the basic EIPSP neuron as PSP is continuously computed, and do not seem to be pertinent to modeling adaptation, fatigue, or facilitation. Adaptation is a rather nonspecific term meaning that the neuron's characteristics adapt to changing conditions. A change in spike threshold should not be entirely ruled out as a mechanism. A change in threshold would represent a change in membrane characteristics, and the real answer to how the observed phenomena are caused probably does involve such a change. But it is more likely that spike production, and not input history, would cause membrane changes at the axon hillock. There is no simple way to envision the spike production/threshold change scheme resulting in the behavior of Fig 6a.

Considerable work was done with a neuron model which changed its threshold in response to inputs. A history of E-inputs would raise the threshold, thus reducing the firing rate. Similarly, a history of I-inputs would lower the threshold. The object here was to make the inputs less potent as they became more numerous, a law of diminishing

returns of sorts. That object was not really met by the method of changing the threshold. The model produced plausible results for E-inputs, and less believable results for I-inputs. Because only a single parameter (spike threshold) changed in order to give the desired effect for both E and I-inputs histories, the algebraic difference (E minus I) was decided upon to apply to threshold change. This is an unrealistic scheme, since no such E minus I input dependence of threshold is present in nature. The results of this model are not included in this thesis.

A more likely scheme is that as inputs arrive at the presynaptic site of neurotransmitter release in great numbers, the neurotransmitter is at first plentiful, but then is depleted, accounting for the loss of potency in the postsynaptic response. Alternately, depletion of the ATP supplies used by the synapse as energy for vessicle release would have the same effect. This would account for the behavior of Fig 6a, but would not explain facilitation, in which a synapse is "primed" by the arrival of a few inputs such that the postsynaptic response grows. It is probable that this behavior results from subsynaptic membrane changes rather than more neurotransmitter made available presynaptically, although the latter has not been ruled out. Fatigue could also result from presynaptic inhibition, wherein outputs are feb back to presynaptic terminals as inhibitory pulses, thus modulating the release of neurotransmitter. Evidence also exists that perhaps repeated outputs cause buildup in the extra-cellular potassium ion concentration, and that this buildup modulates the release of neurotransmitter [Ref 16].

Fortunately, it is unnecessary to chose one or the other when modeling, since only the postsynaptic response is of concern. PSPFAT, then, varies the potency of E and I-inputs by varying the size of the postsynaptic response acccrding

to the running average of inputs. Thus the excitatory and inhibitory effects can be handled separately. As a mass of E or I-inputs occurs, the size of the postsynaptic response decreases, thus modeling fatigue.

In the PSPFAT program, this task is handled by counting inputs over the specified time interval preceeding each word of the 1K block.

Since only integer arithmetic is used in neural modeling (in order to maximize core usage), a small reduction in the postsynaptic response strength of say one-tenth had to be handled indirectly. This was done by first dividing both the E and I postsynaptic response blocks by 128, and then computing (on each pass) an integer for building them back up for PSP formation. These integer multipliers take into account the running averages and the factor by which the running averages affect the postsynaptic responses. Specifically, the integer for building the PSP blocks back up is [128-(factor X running average)]. If the factor is positive, the integer will be smaller than 128, and fatigue results. By selecting a negative factor, synaptic facilitation can be modeled.

F. LATERAL INHIBITION

The original lateral inhibition program is a highly complicated, intricate, and interactive composition, and is a great credit to LT. Dennis Marvel, who wrote it with suggestions from Dr. Marmont. When the author inherited it, however, it had several shortcomings, which will be enumerated as they occur.

LINHIB consists of eight 1K data blocks, each

representing a channel, or neuron, such as a retinal ganglion cell (Fig 7). LINHIB models any lateral inhibition network, however, and is not necessarily retinal. Inputs to the network are from upstream neurons (perhaps bipolar cells) which synapse on each of the eight ganglion cells. Outputs are sent via the optic nerve to the brain for further processing. Inhibition results whenever an output occurs. An output from a neuron inhibits its neighbors, but never itself, and is mediated by amacrine and horizontal cells in the retina.

The original version of LINHIB produced equal inhibition in all seven neurons each time an output occurred. This restraint was removed by the addition of program segments which compute the separation between the neuron producing the output and the neuron being inhibited, and then perform a table lock-up to determine the appropriate inhibitory strength. Termed the lateral inhibition decay (LID) table, a typical entry would be 5432100. The first number describes the strength of inhibition of the neuron adjacent to where the output occurred, while the last number describes the strength of inhibition of the neuron most remote from where the output occurred. Therefore, this LID describes decay in relative inhibitory strength as distance from the output neuron increases. Later, as more flexibility was desired, a two sided LID table was devised, allowing inhibitory influence to decay differently in different directions. A typical two-sided (but symmetrical) LID table might be 001234505432100, with the central zero always present to indicate no self-inhibition. Thus LID=000000005432100 would produce a network in which inhibition occurred to one side only.

Originally, all inhibition occurred after a fixed delay in time (or really, words of the block). In order to add more flexibility, this was changed to allow a linear delay

in the spread of inhibition. That is, if the neuron adjacent to the output is inhibited after 0.001 seconds, then the next would be inhibited after 0.002 seconds, the next after 0.003 seconds, and so on (Fig 8). This linear delay could be accomplished through synaptic delays, as shown.

The LINHIB program works in much the same way as the PSP production in the basic EIPSP model. That is, the EPSR and the IPSR are generated in a pair of 1K blocks which are aged and summed the proper number of times to form each PSP word. The biggest difference is in the way this is done. Because LINHIB models eight neurons vice a single neuron, much more memory is required. It would have been impossible to fit eight E-input blocks, eight I-input blocks, eight output blocks, eight PSP blocks, and two PSR blocks all into the core available. Therefore, the E-inputs, outputs, and I-inputs are all stored in compressed form. The E-input and output blocks are each 256 word blocks containing the addresses of the markers. The I-inputs are stored in a 2K block where a given inhibitory unit consists of one word of address and one word of relative strength.

Processing begins by stepping through word number one of each of the eight data blocks, and polling the compressed E-input block to find an address match, which, if found, signals an E-input at that location (Fig 9). Each time an E-input is found, a zero is placed on the E-age stack. The zero indicates that the PSR has not aged at all, since the input has just occurred. Also, an E-input counter is incremented. Next, the PSP word is formed by adding the proper number of properly aged PSR's to the PSP block. When word number one of all eight data blocks is processed, a similar procedure is followed for inhibitory inputs. The difference here is that the compressed I block is different in format; the I address is compared with the current processing address to detect a match. Then, the inhibitory

strength is used to increment the I address for more matching. Thus if the relative inhibitory strength is five, this would have the effect of producing five inhibitory pulses.

Following the subtraction of the proper number of properly aged IPSR's from the PSP word in each of the eight blocks, a comparison of PSP with spike threshold is made. If PSP equals or exceeds spike threshold, the proper address is entered in the compressed output block, and the task of placing inhibition is begun (this has already been described). Finally, processing steps to the next word and returns to the top of the flow graph.

After much work had been done with the LINHIB program, the unsettling discovery was made that many of the results were probably invalid because of overflowing the capacity of the stack which is used to age the inhibitory inputs. As mentioned earlier, when graded inhibition was added to the program, this was done by providing more markers for stronger inhibition, and fewer markers for weaker inhibition. The actual inhibitory effect was then set by adjusting the value of the IPSR. The end result of multiple markers was to fill and in some cases overflow the stack, thus invalidating many earlier runs. This problem was solved by enlarging the E and I-age factor stacks from 1K to 6K in size. The extra core memory was gained by rewriting the entire program in the APTEC "language." Additionally, program steps were added which check the compressed E-input, I-input, and output blocks, and E/I-age stacks for overflow, and warn the operator of these undesirable conditions.

Another point of concern which turned out to be entirely cosmetic involved the placement of inhibition after the ramp delay was added. Because the eight processing blocks are continuous in core, an output near the end of one block will

produce inhibition at addresses which are beyond the end of the block intended, but which fall near the beginning of the following block. It turned out that these bogus I-pulses do not get taken as inputs for PSP computation, because by the time they get placed in the compressed I-block, processing is well beyond their location. They do get displayed, however, but this problem is easily solved by graphical methods.

G. INLIB AND OUTLIB

The various 1K blocks of input pulse trains are built by a program called INLIB. The operator selects one of two versions of random generation, one of two versions of constant frequency, or frequency modulation. These are stored on disk uncompressed for use with any program except LINHIB, which requires a 256 word compressed E-input block. The formation of this compressed block for LINHIB is by a program called OUTLIB, which reads any eight 1K blocks from disk and compresses them by storing marker addresses in the 256 word block which is then stored on disk. The author rewrote these programs in APTEC, improved the frequency modulation program's flexibility, wrote one new program for generating constant frequency blocks, made program modifications which increased the total number of inputs which could be placed in the 256 word block, and generated numerous additions to the existing library of inputs.

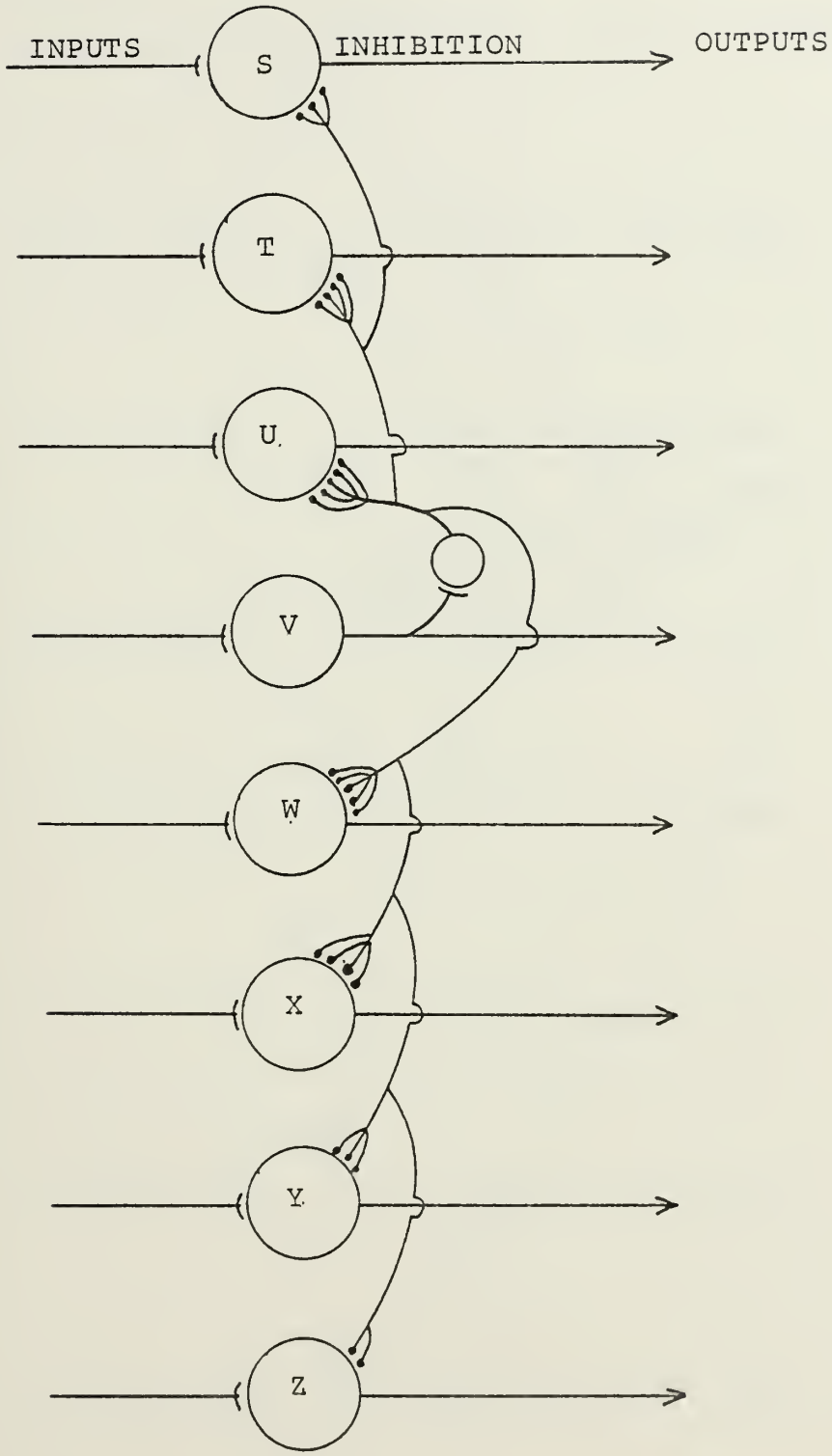


FIGURE 7 - LINHIB NETWORK, CONSTANT DELAY

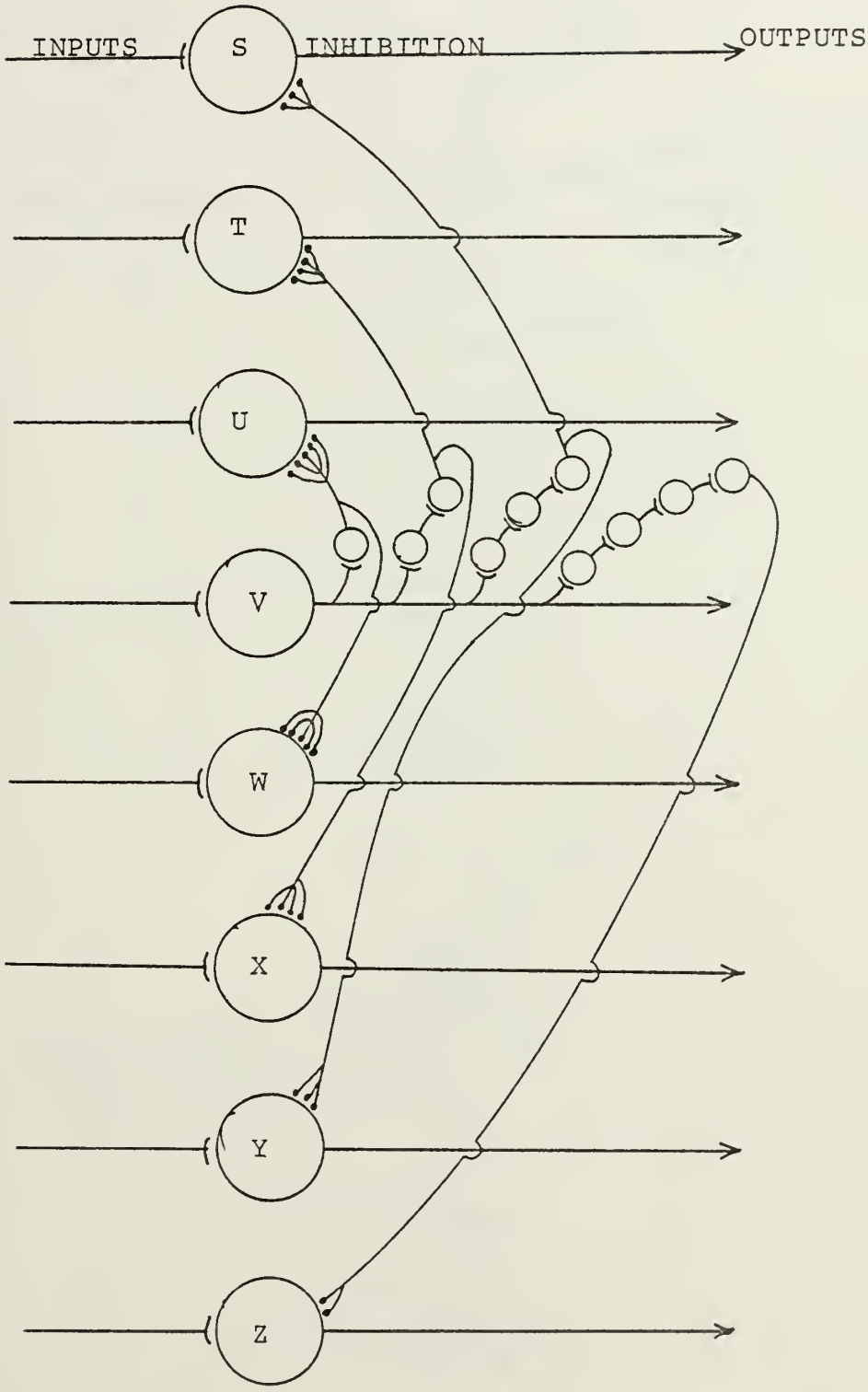


FIGURE 8 - LINHIB NETWORK, RAMP DELAY

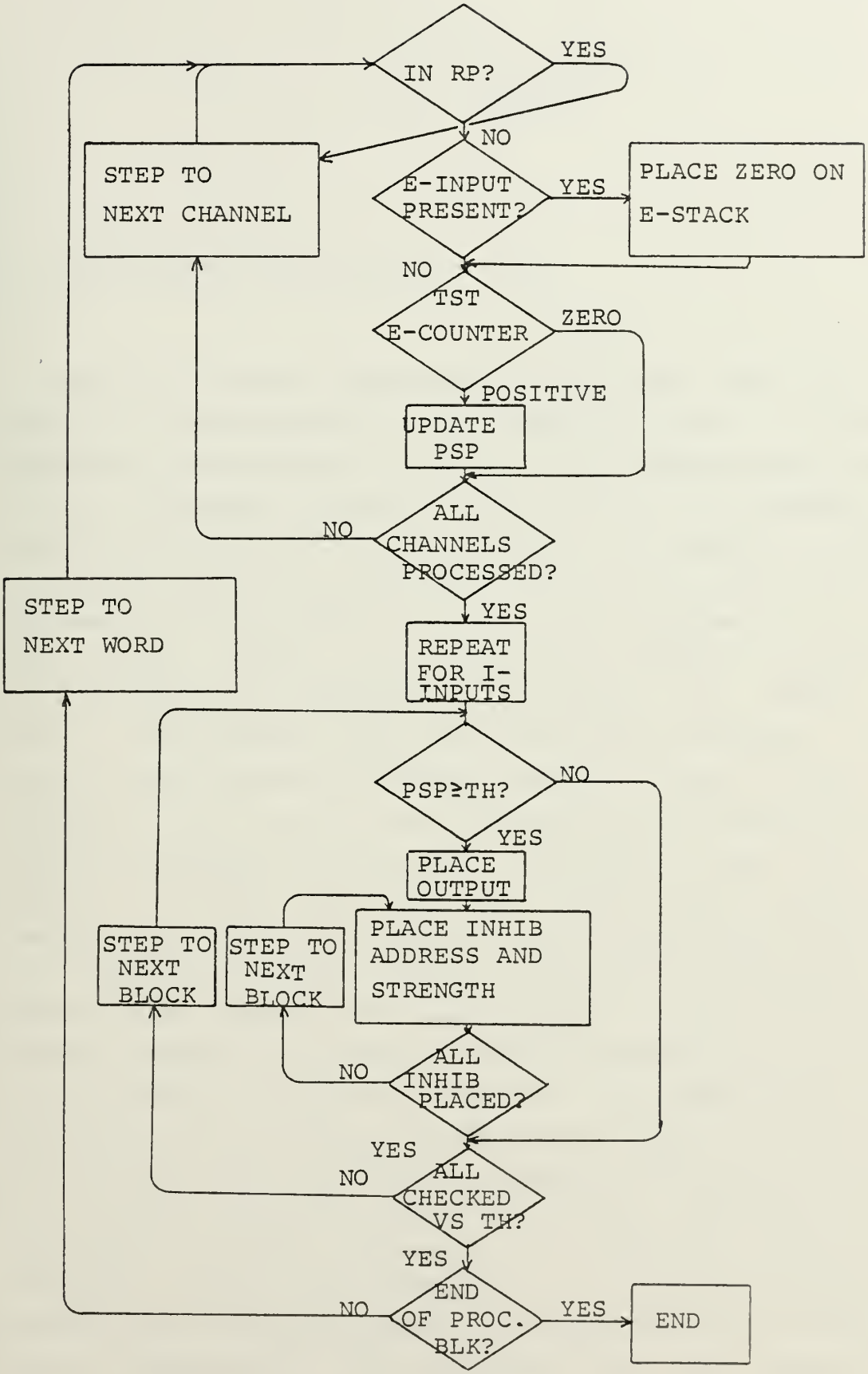


FIGURE 9 - FLOW CHART OF LINHIB

IV. RESULTS

A. CONVENTIONS FOR PLOTTING OF DATA

There are several clarifying points which will aid the reader in understanding the modeling plots. 1) Should excitatory, inhibitory, or output spike markers appear which are of differing heights, this is of no account. The programs simply test for the presence of a non-zero marker, not caring about its magnitude. 2) Not all plots were made for every run for LINHIB, as this would require two or three full pages. Rather, only those plots needed to illustrate the discussion are included. In general, the PSR's, the PSP's, the inputs, and the outputs are included. 3) The sizes of the IPSR and the EPSR relative to each other are accurate, but due to the consideration of making as much detail as possible in the space available, they will not always be scaled the same as the PSP plot. Scaling is, however, done after all computations are complete, and in no way affects the accuracy of the results. 4) The plots each cover a time course of 100 milliseconds. 5) The difference between threshold and resting membrane potential is taken to be 20 millivolts. This can be gauged by noting that upon occurrence of an output, the PSP trace drops vertically from threshold to rest potential and remains there for at least the duration of the refractory period. All plots use 20 millivolts as spike threshold. 6) Multiple markers in inhibitory plots are understood to be an indication of strength of inhibition, and not actually multiple inhibitory action potentials. (7) All plots are the results of

computer calculations made with the modeling programs described. The plots are therefore not merely how the author thinks things should happen, but how computer calculations indicate they should happen.

B. TEMPORAL PHENOMENA

It is impossible to really separate "spatial" from "temporal" when dealing with a spatial neural array processing a time sequence of events, but the distinction drawn here is just as the reader might expect: those situations in which a change in time are predominantly important are covered under temporal, whereas if the change from neuron to neuron is most important, it is termed spatial.

1. Fatigue and Facilitation

The first temporal phenomena to be modeled are fatigue and facilitation.

Figure 10 shows a neuron, initially quiescent, receive a train of E-inputs. As the E-input history increases, the PSR due to each E-input becomes smaller. This can be seen by observing the PSP trace carefully. At first each E-input produces a sizeable response. But as E-inputs accumulate, the PSR's become so small that they are barely perceptable atop the quiescent ramp. As discussed earlier, this behavior probably results either from neurotransmitter depletion or subsynaptic membrane changes. Figure 11 shows fatigue in the case of I-inputs. The output trace clearly shows an initial extinction and a gradual return to firing at just below the quiescent rate.

By selecting a negative number for the fatigue factor in PSPFAT, facilitation can be shown (Fig 12). Initially quiescent, the neuron is excited by the now familiar E-input train. As E-inputs accumulate, some submicroscopic effect causes the PSR for each to wax in strength, resulting in an accelerating output. The facilitation modeled here is of very short duration, and should not be confused with any long term changes which result from the plastic nature of synapses [Ref 7]. Such long term synaptic facilitation is a likely mechanism for long term memory. The very short duration facilitation of Fig 12 could conceivably be responsible for a person being able to glance at a table full of objects, look away, and picture the scene, enabling him to name the objects, whereas after several seconds, this is no longer possible.

One uncertainty in the fatigue models is that the real-life phenomenon might have resulted from receptor adaptation rather than from synaptic changes or subsynaptic membrane changes. The E or I-input train used in the models was, in most experimental measurements, actually a light dot or surround stimulus rather than a known pulse train of constant frequency. Therefore, if a constant intensity light stimulus had resulted in receptor adaptation, the proper representation of inputs to the neuron would be a train of decreasing frequency. This alone could account for the decreasing output rate after the stimulus had been on for a short time. The light and dark adaptation of receptor cells is a well known phenomenon, but this occurs with a time constant on the order of minutes, and could not result in changes on the order of one second or less. A review of individual receptor responses in the frog retina to light stimuli [Ref 15] indicates no discernable adaptation over a period of one second. This may not be true of all retinal receptors, however.

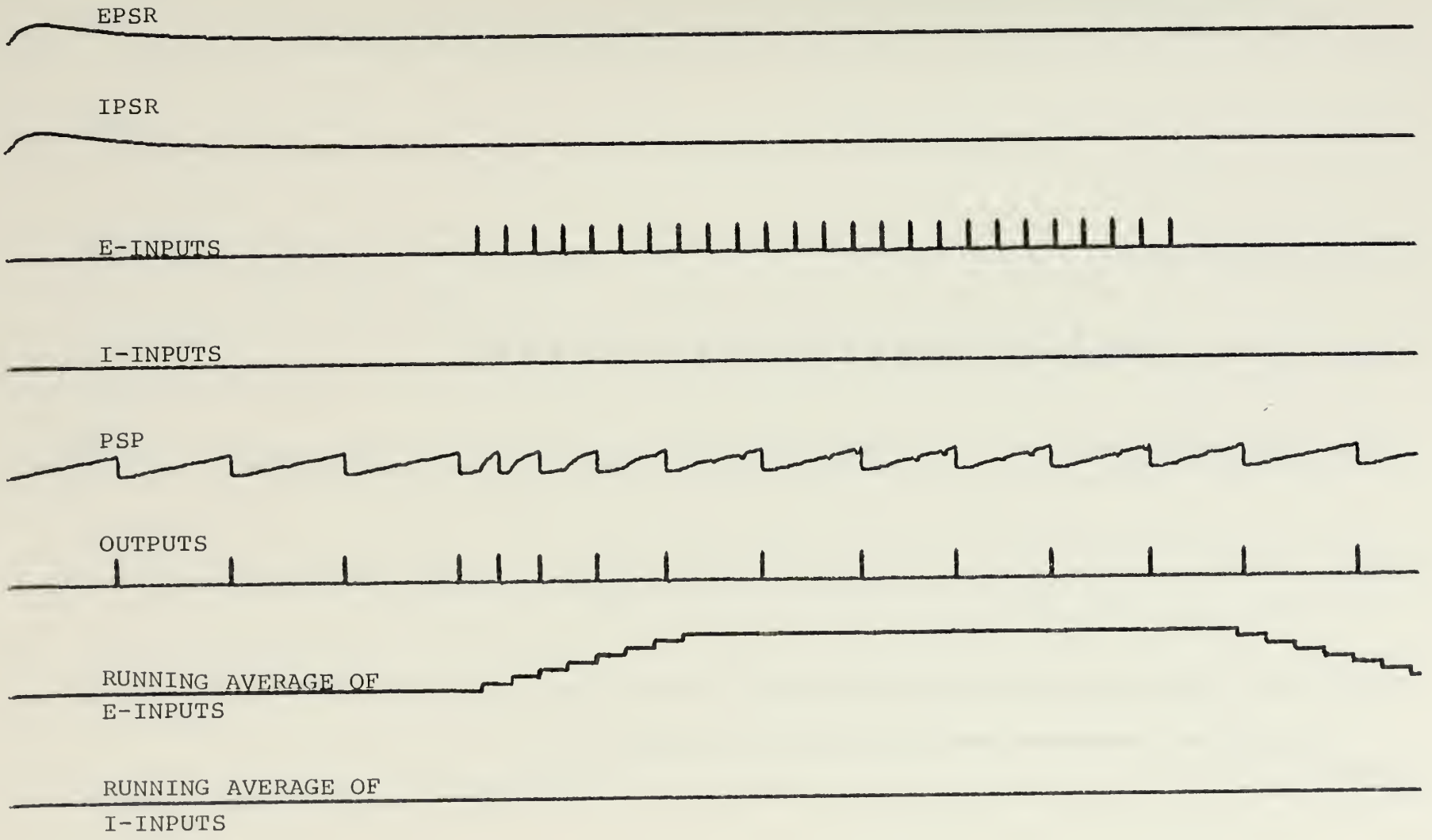


FIGURE 10 - FATIGUE OF E-INPUTS

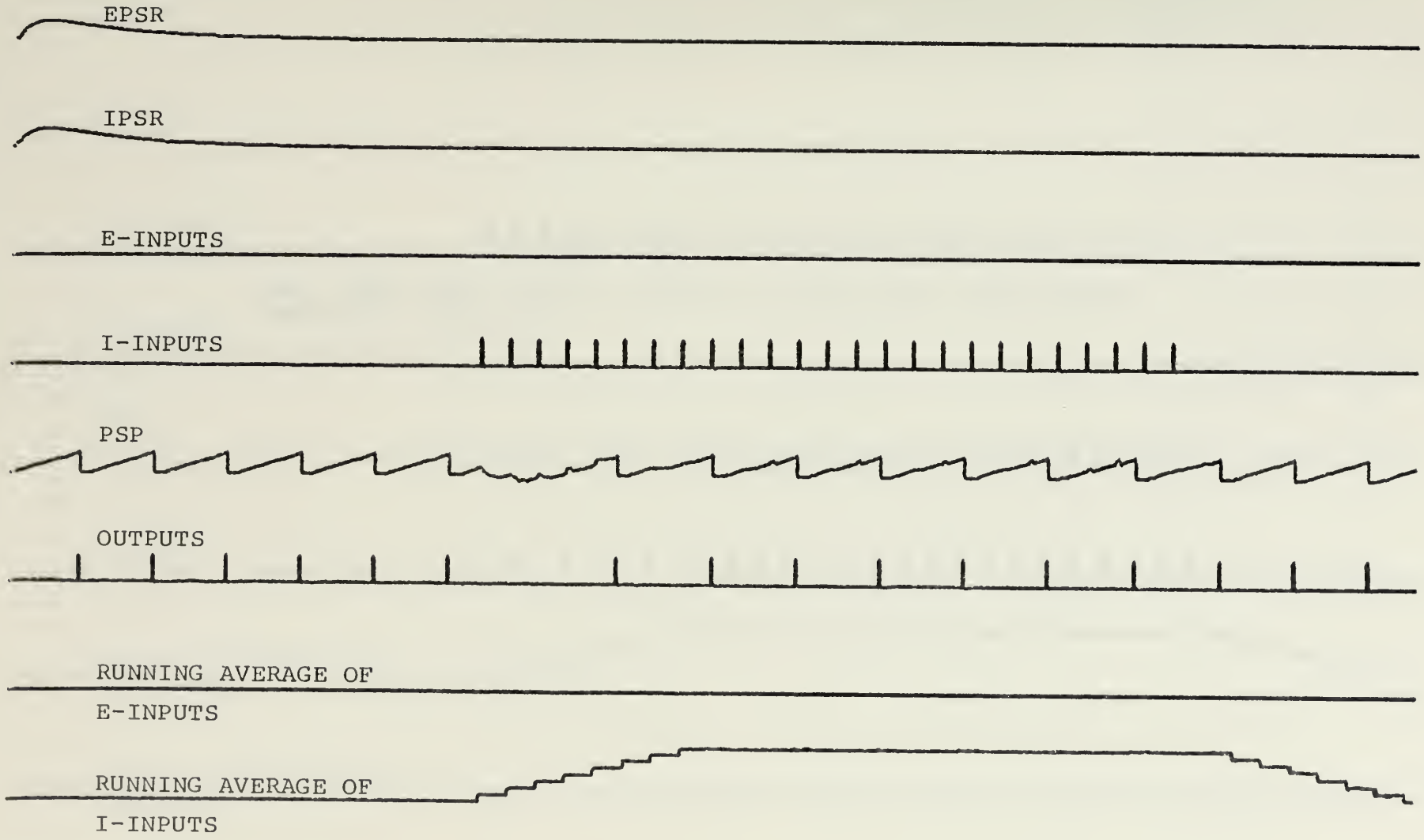


FIGURE 11 - FATIGUE OF I-INPUTS

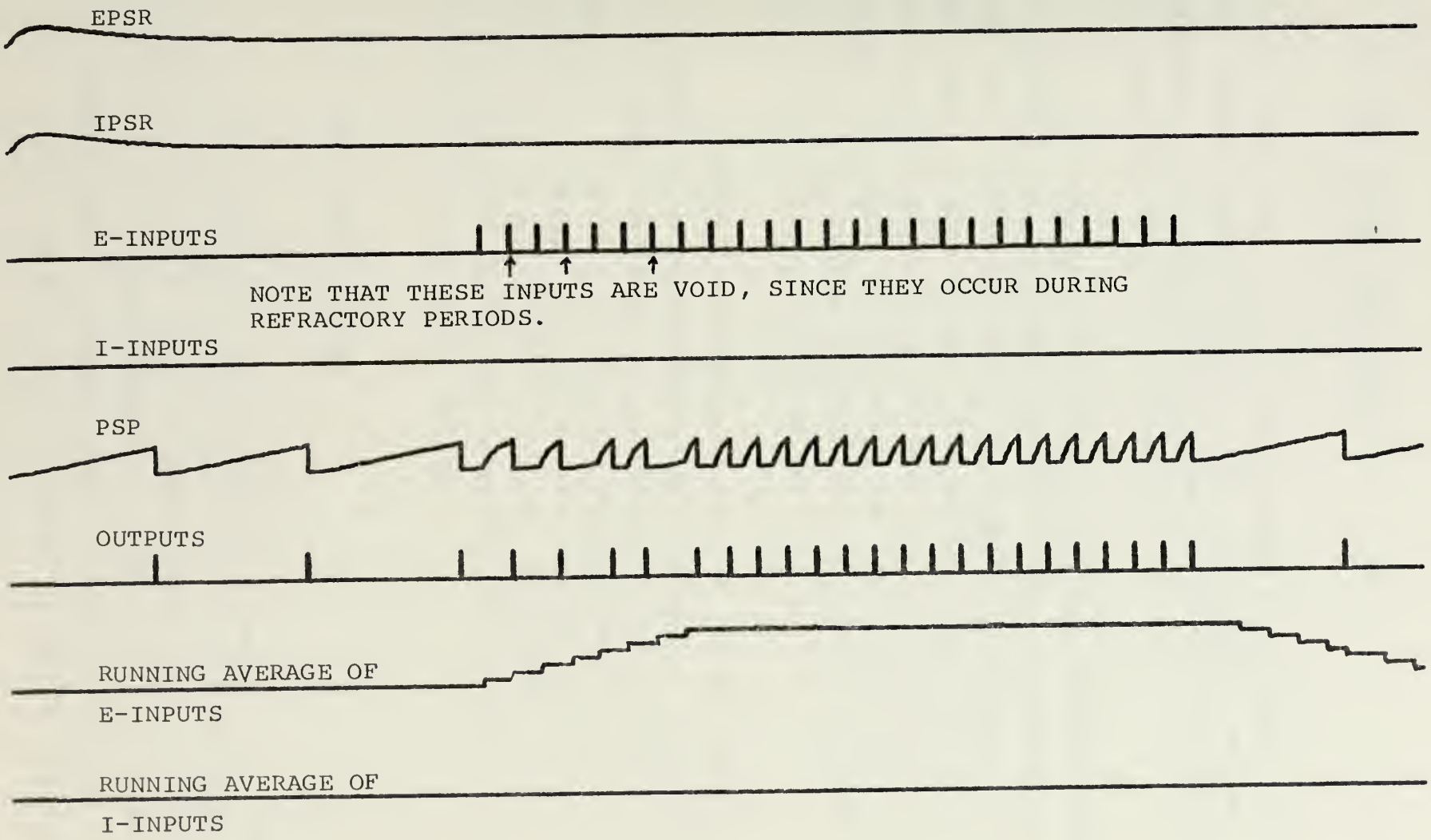


FIGURE 12 - FACILITATION

2. The Neural Oscillator

The neural oscillator is used to generate bunched E and I-pulse trains for use with the target neuron. The character of these bunched pulse trains can be observed in the E and I-input plots of Fig 13b.

One mode of generating the required outputs consists of allowing the oscillator neuron to have a quiescent firing rate, and then feeding back the outputs as inhibitory inputs (Fig 13a). By then using the output and feedback pulse trains as driving inputs to neuron B, the oscillatory PSP is created (Fig 13b). Alternately, the oscillator neuron can be provided with random E-inputs (Fig 14a). When outputs occur they are fed back as I-inputs, as before.

Although instability in the oscillator neuron is not of importance to other work in this thesis, a demonstration is included to show the versatility of the NEUOSC program (Fig 15). Note that originally, outputs result only from the quiescent firing rate. Since the outputs are fed back as E vice I-inputs, the outputs wax in intensity at an ever increasing rate until the neuron fires at near the maximum allowed by the refractory period.

3. The Target Neuron

The outputs of the oscillator neuron are used to create a sinusoidally varying PSP in the target neuron. The target neuron would then fire most strongly in response to E-inputs at the proper phase and frequency. Conversely, if the E-inputs were of random character, the target neuron could produce a very regular output of known frequency and

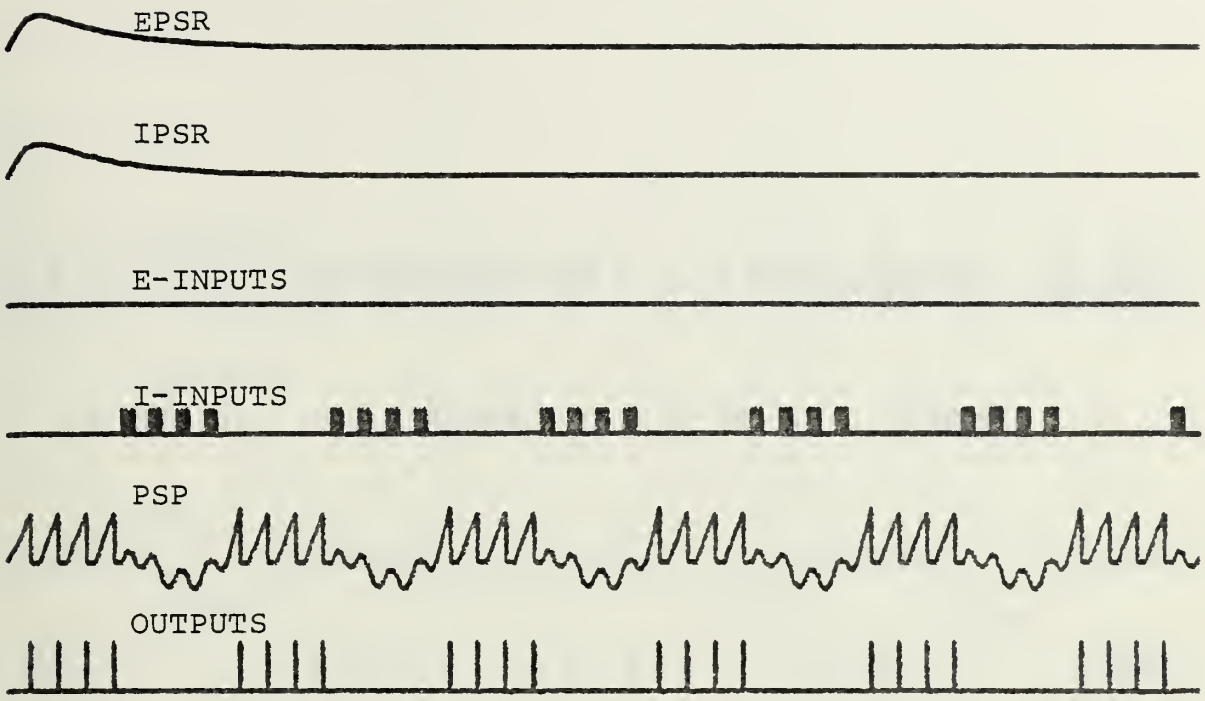
phase.

A good example of a higher frequency sinusoidal PSP is shown in Fig 16. Note the role of alternating E and I-inputs in driving the PSP in opposite directions. If an oscillating PSP were created using E-inputs alone, the slow decrease of the PSR would cause a gradual depolarization toward spike threshold, placing a severe upper limit on the frequencies and amplitudes attainable. The idea that higher frequencies may be used in CNS processing is being pursued in electroencephalogram work by Dr. Marmont and his students in the 70-95 hertz range. It is probably an insult to the CNS to believe it incapable of useful activity at frequencies above ten hertz [Ref 11].

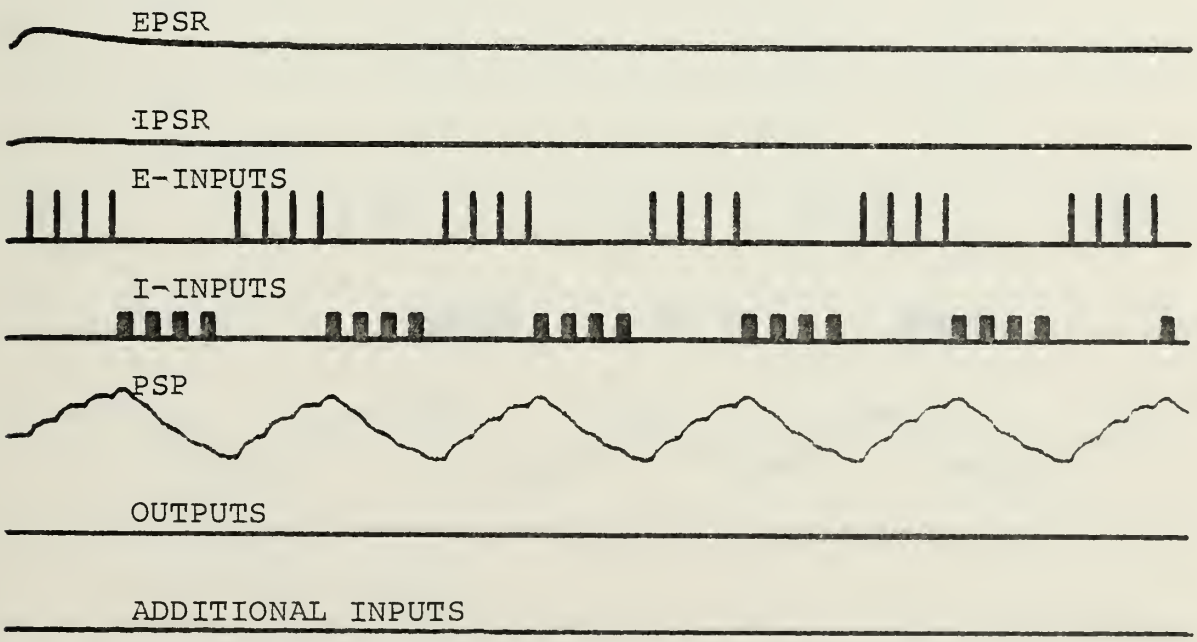
Figure 17 is an example of regularization of random inputs. A basic constraint is that the PSP driving inputs should be much more numerous than the random inputs, or the latter will tend to dominate the PSP characteristics. Alternately, providing a weaker PSR for the random inputs would allow the driving inputs to dominate the PSP, even though the random inputs might be very numerous.

4. Motion Detection

Motion detection systems have been demonstrated by microelectrode studies in many species. These studies tend to indicate that motion detection is performed by neurons having very specific characteristics as to velocity, direction, and form for optimum detection. The adaptive advantage of an efficient system for motion detection is obvious: the objects in the visual field which are of greatest interest to an animal in the wild are those which are in motion.

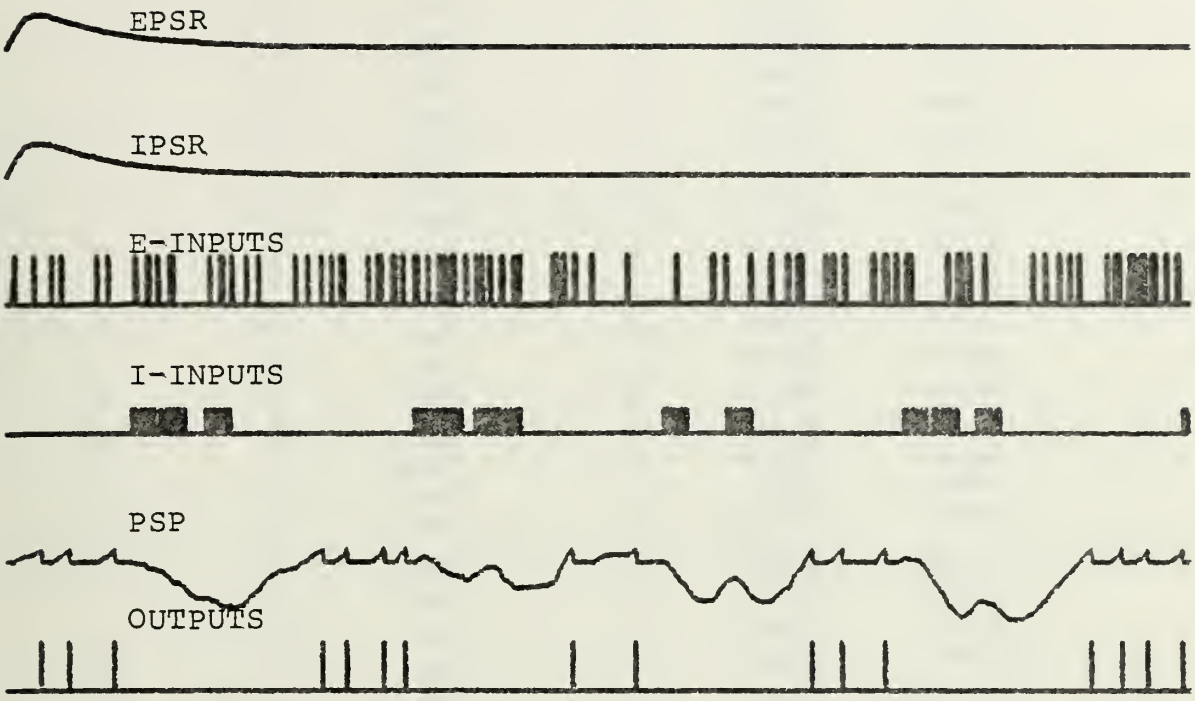


A. OSCILLATOR NEURON

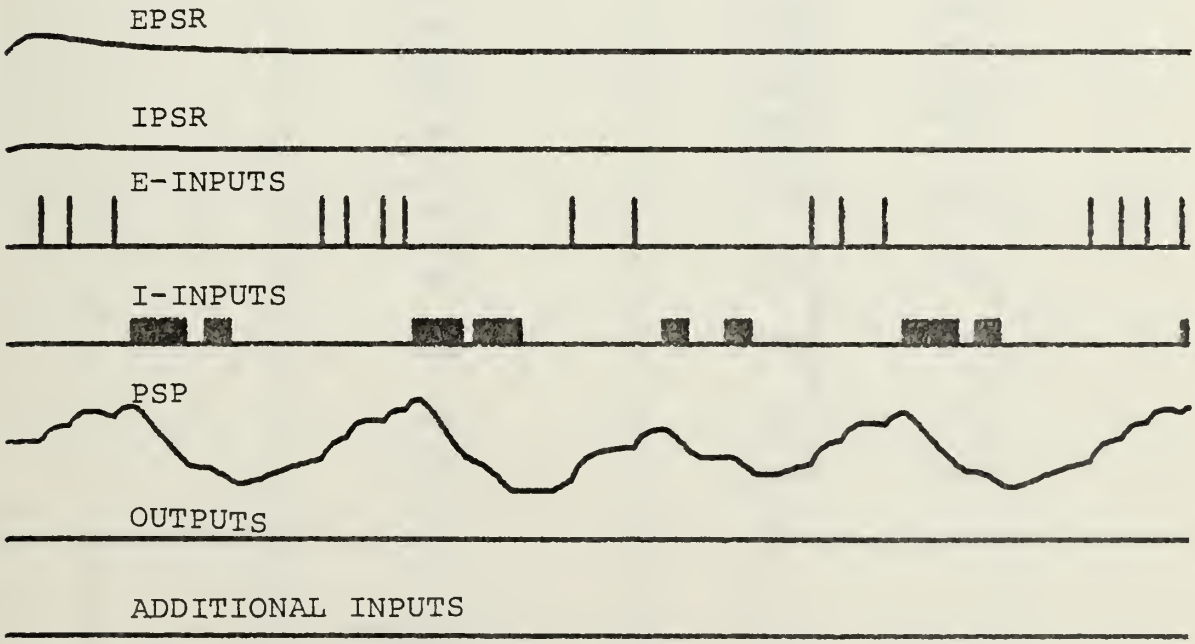


B. TARGET NEURON

FIGURE 13 - SINUSOIDAL PSP, SPONTANEOUS



A. OSCILLATOR NEURON



B. TARGET NEURON

FIGURE 14 - SINUSOIDAL PSP, RANDOM INPUTS

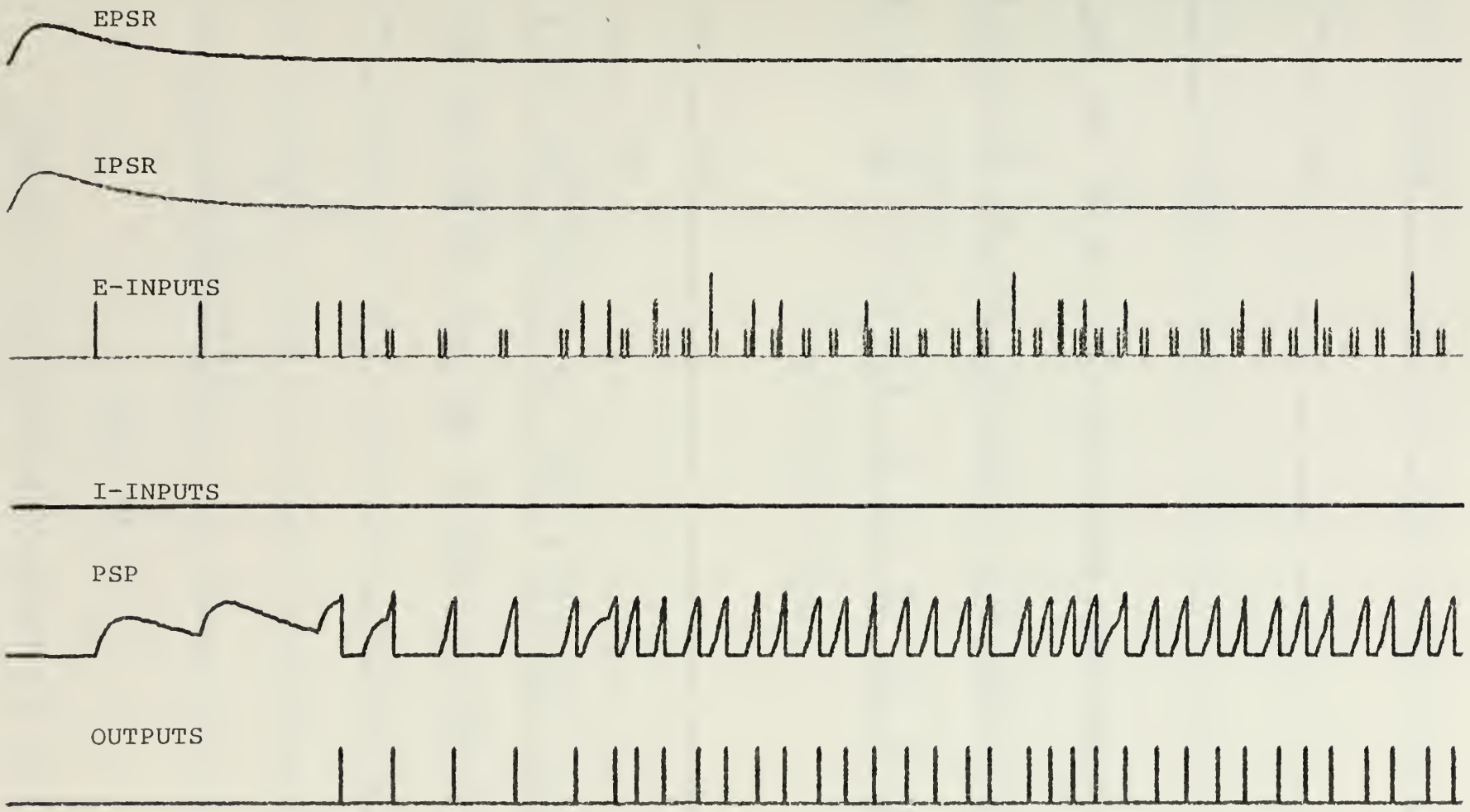
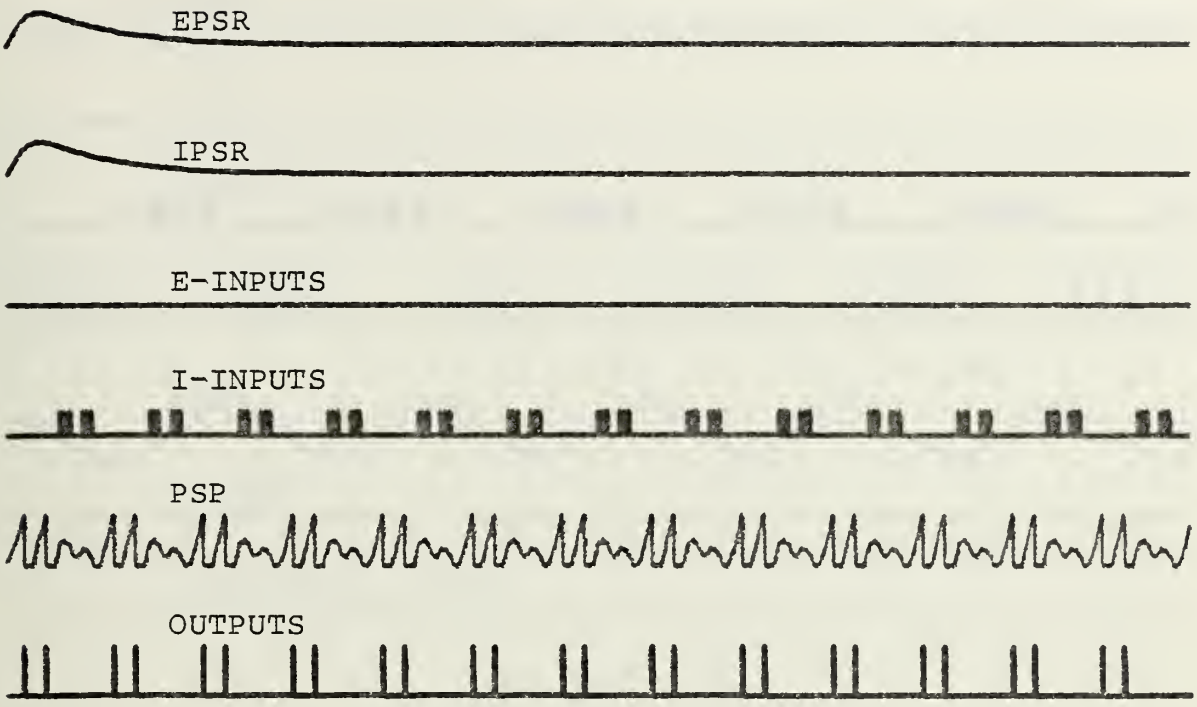
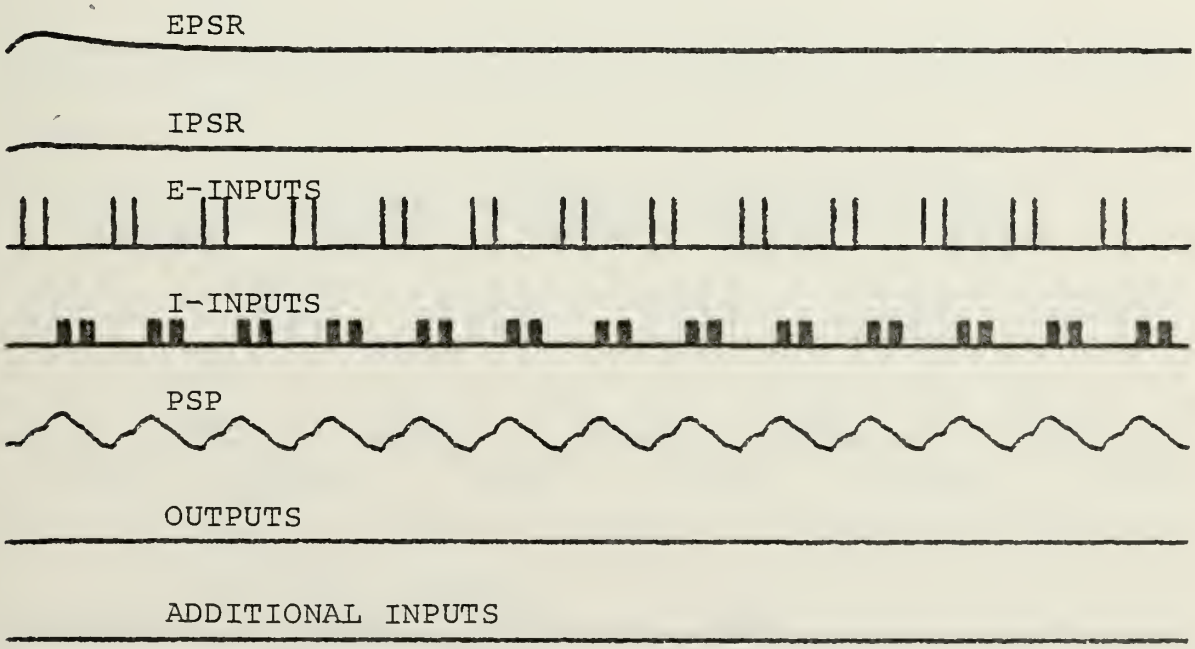


FIGURE 15 - INSTABILITY

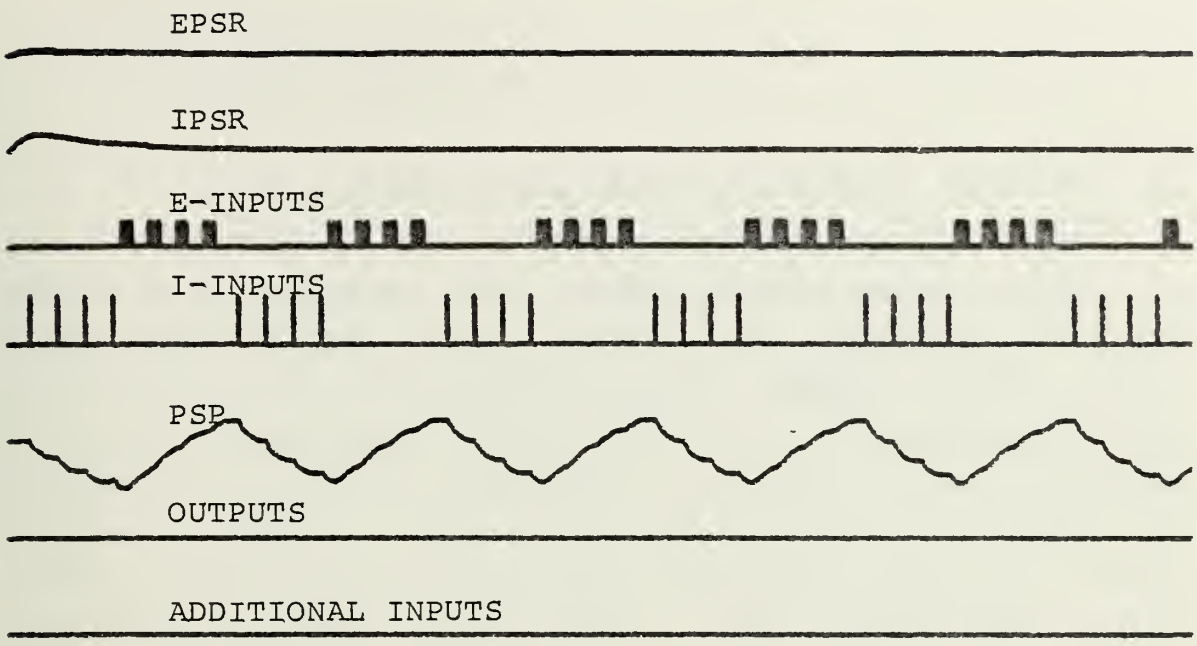


A. OSCILLATOR NEURON

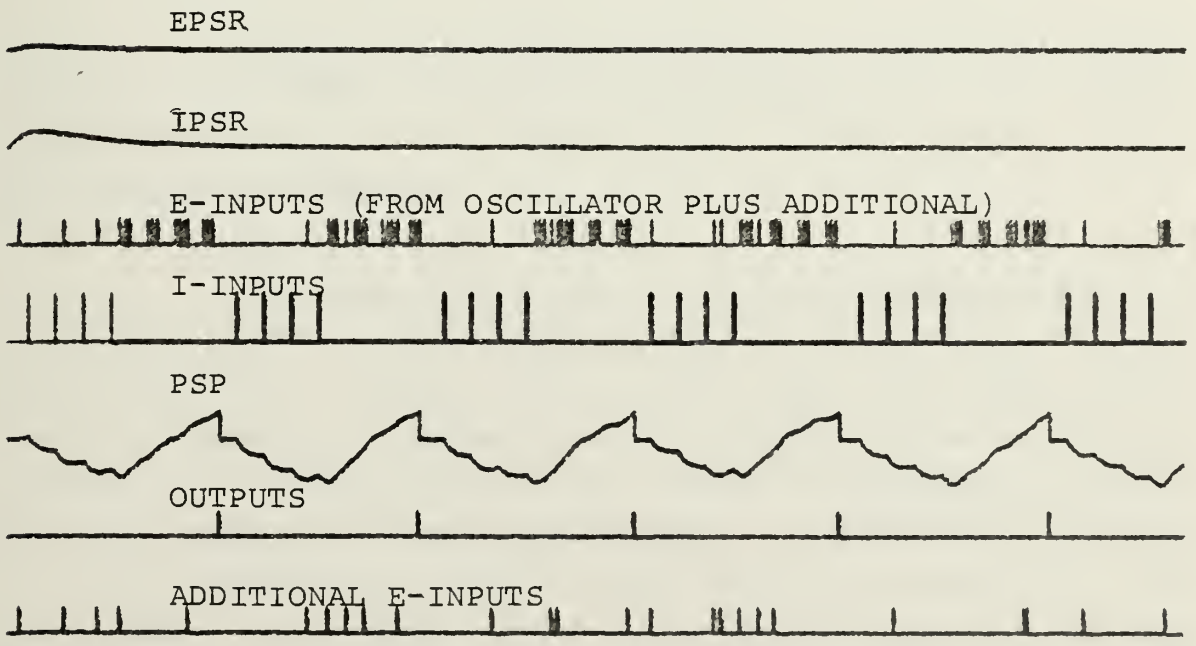


B. TARGET NEURON

FIGURE 16 - HIGH FREQUENCY SINUSOIDAL PSP



A. OSCILLATOR NEURON



B. TARGET NEURON

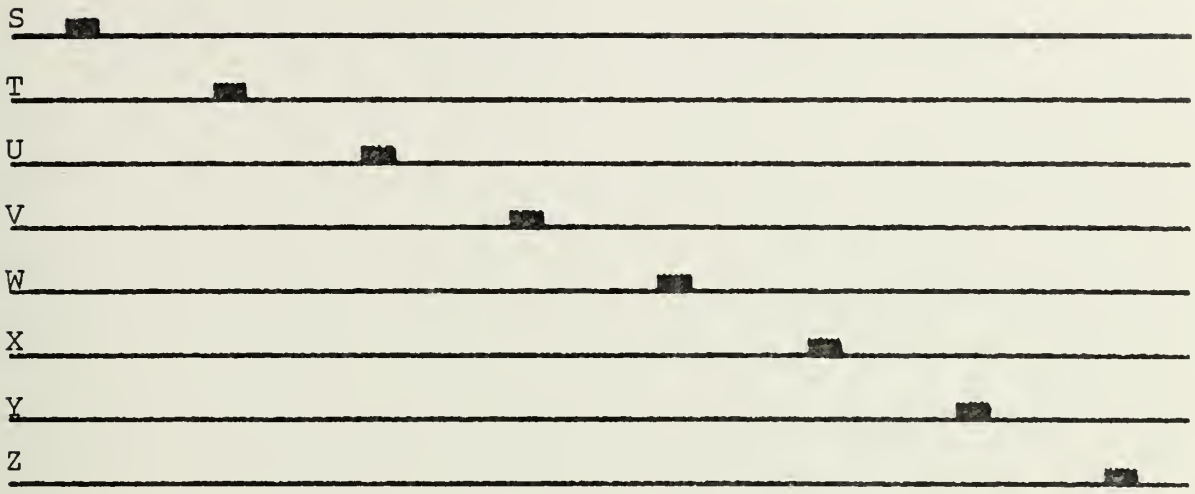
FIGURE 17 - REGULARIZATION BY SINUSOIDAL PSP

a. Band Pass

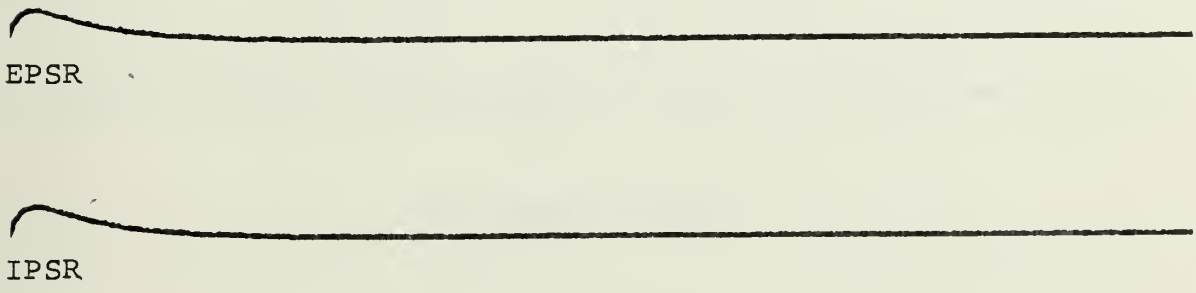
The target neuron with oscillatory PSP can be put to practical use as a target velocity band pass gate. A tiny target crossing the LINHIB eight neuron array is represented by successive inputs at each neuron (Figs 18 and 19). The outputs are applied to the target neuron, where the PSP is oscillating (Fig 20b). Here, only E-inputs from the neural oscillator (Fig 20a) have been used to create the proper frequency. Because the inputs to neuron B from the LINHIB network are at the proper frequency and phase, strong outputs are produced from neuron B. This signal might say to the cortical centers for visual association that there is a target in the receptive field of neuron B which has X radians per second of angular velocity. The organism could then take action appropriate to the stimulus.

Neuron B is a summing neuron, since all eight channels of LINHIB converge to excite neuron B. The summing is performed only if the inputs occur at the proper points on the PSP oscillation.

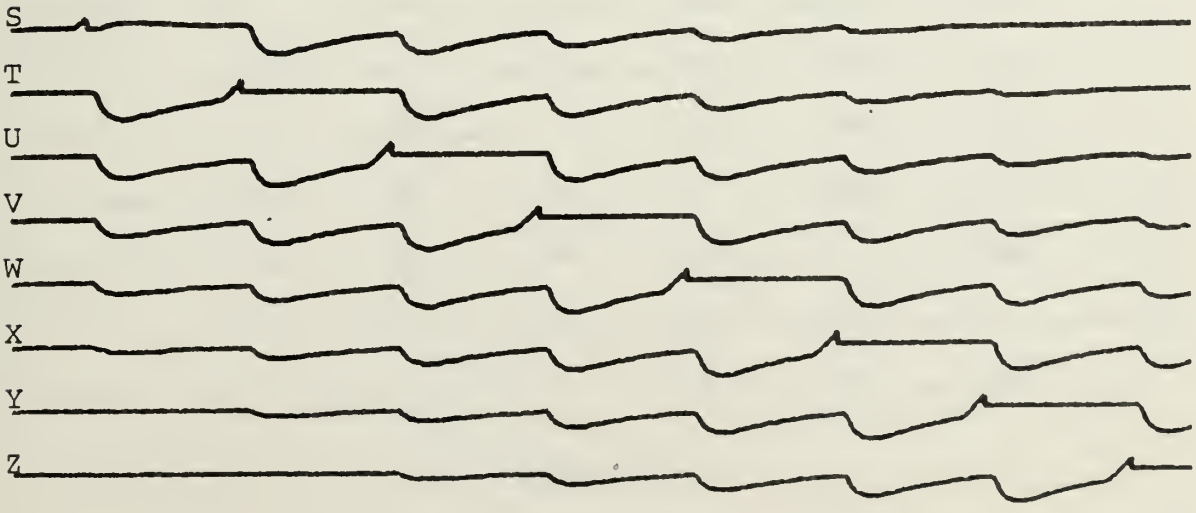
The fact that phase as well as frequency must be correct in order for the speed band pass gate neuron to pass the signal is a sticky point which has not been fully solved. Perhaps the appearance of a target causes a strobing effect in the oscillating neuron which automatically adjusts the PSP oscillations to optimum phase. Alternately, perhaps a given frequency has four or more target neurons such that any phase of target would cause outputs from one or more target neurons. Creating multiple target neurons with oscillatory PSP's of different phase is a simple matter of delaying the driving E and I-input pulse trains by a fraction of the oscillatory period, as shown in Fig 2b.



A. INPUT

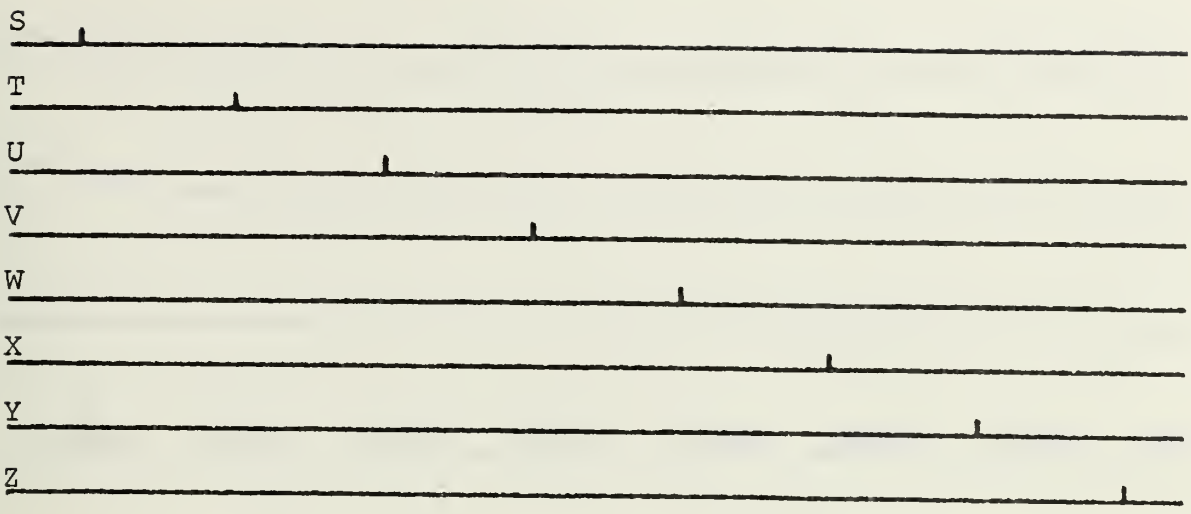


B. PSR



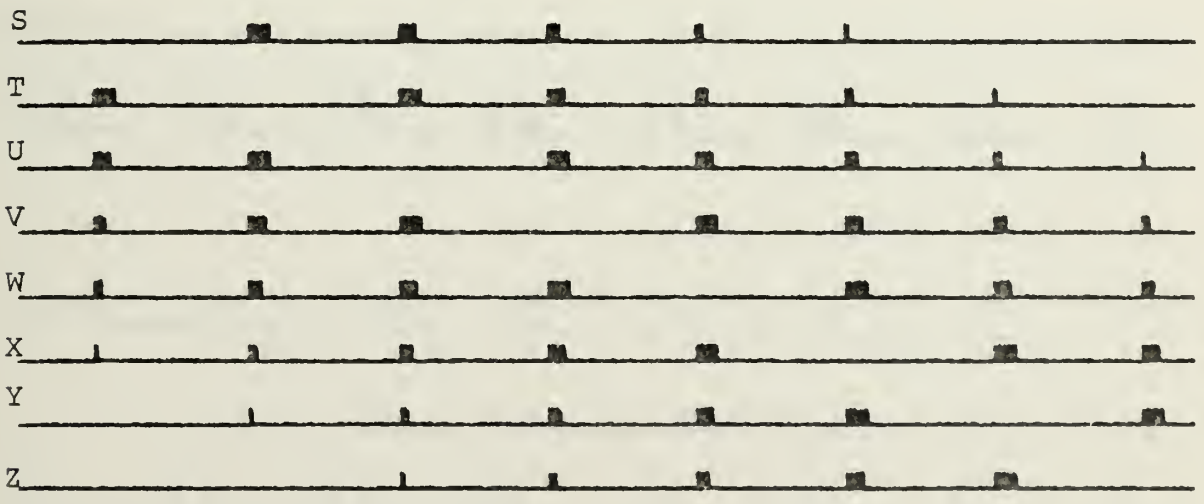
C. PSP

FIGURE 18 - LINHIB WITH DOWNWARD TARGET, PART 1



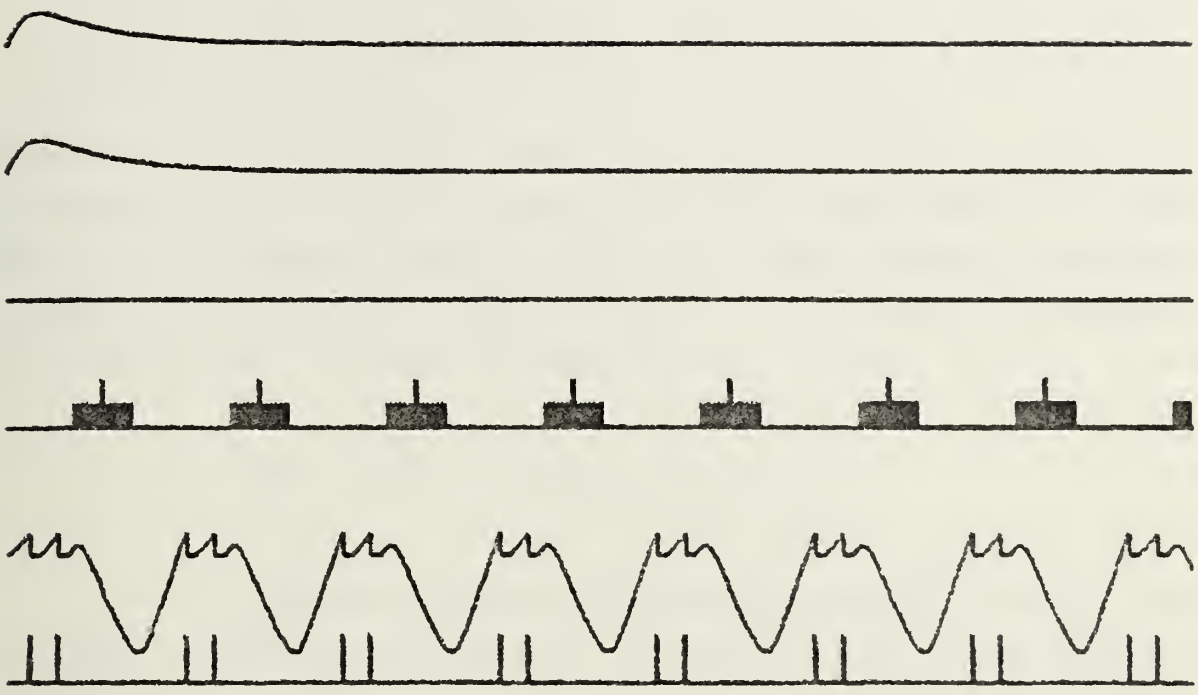
A. OUTPUT

B. SUMMED OUTPUT

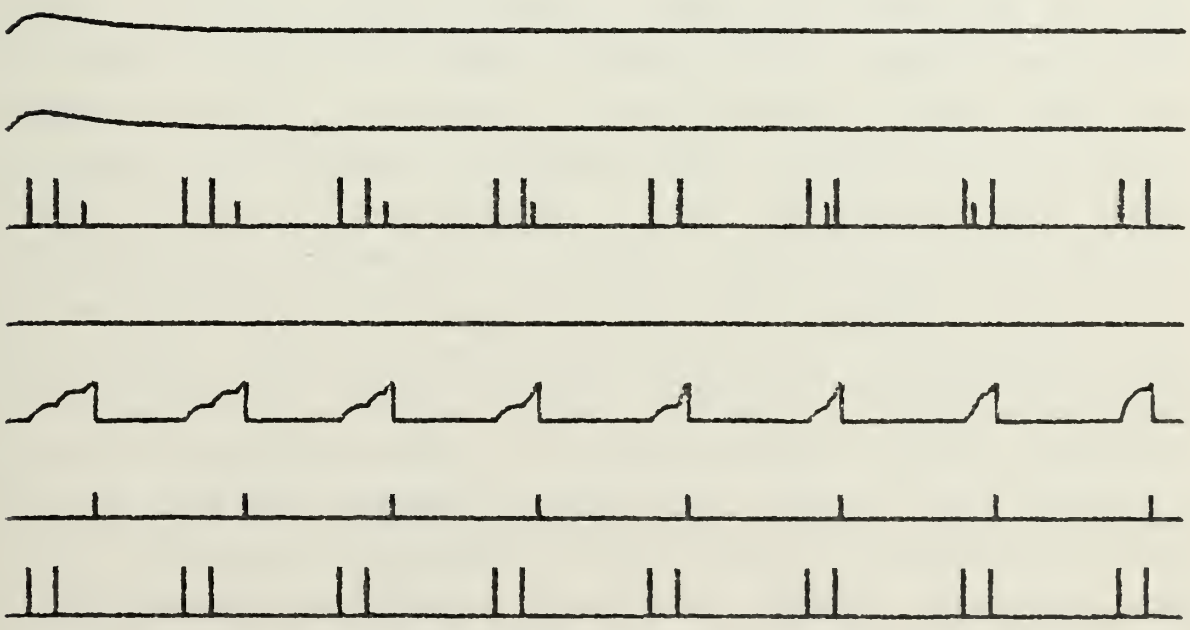


C. INHIBITION

FIGURE 19 - LINHIB WITH DOWNWARD TARGET, PART 2



A. OSCILLATOR NEURON



B. TARGET NEURON

FIGURE 20 - SPEED BAND PASS DETECTOR

There is evidence that neurons do exist which are specific for speed of target motion [Refs 1 and 8], as well as for direction. Pontine cells respond to a band of speeds, whereas retinal speed specificity tends to be less discrete than a narrow band pass gate might provide. That is, in the retinal ganglion cells of the rabbit, response tended to be broken down into two crude groups: high speed detectors and low speed detectors.

b. Null versus Preferred Direction

Direction specificity is now modeled using the lateral inhibition network. In Figs 18 and 19 the lateral inhibition network was used, but the parameters were adjusted so that the lateral nature of the network was suppressed. In other words, neuron S has an early output, and produces inhibition in its neighbors. But this inhibition is no longer present (PSP has returned to the resting value) in neuron T when E-inputs representing the target reach it. Obviously, if the duration of the PSR for I-inputs were longer, a moving target could be blanked out. Furthermore, if inhibition in one direction were much stronger than inhibition in another direction, target motion in one direction could be detected while target motion in the other direction would be blanked out. Figures 21, 22, and 23 show the results of this modeling. Since the LID table is 000000005432100, the upward target is not inhibited at all, and each neuron produces an output. The downward target, however, produces only two outputs, in neurons S and X. When neuron S fires, inhibition spreads downward and hyperpolarizes the membranes of the neurons below it. Because a long duration IPSR was used, outputs are prevented in the four neurons below neuron S. Neuron X is both later in time, and had weaker inhibition to begin with, so it

produces an output when excited. As with neuron S, the output from neuron X is effective in preventing outputs from neurons Y and Z. Thus upward is the so-called preferred direction, while downward is the null direction.

The target neuron is once again a summer. For all circuits except band pass, it is a simple summer, where one input is generally sufficient to produce an output. For band pass, frequency and phase of inputs must match frequency and phase of the oscillatory PSP in order for potent summing to occur.

There are important differences between this model for null versus preferred directions and that of Ref 12 (Fig 24). The density of receptors in the retina evolved to a very high value for very good reasons, namely sensitivity and acuity. More receptors means that many receptors can (via bipolar cells) feed a single ganglion cell, resulting in very good sensitivity. More receptors also means that visual acuity improves, sensitivity being constant. The neural circuit of Fig 24, ignoring the dashed connections, has the disadvantage of using precious receptors for a dedicated purpose. This would degrade sensitivity and acuity, receptor density held constant. The model used in this thesis, on the other hand, has all receptors synapsing in the "normal" manner, and uses lateral inhibition among the ganglion cells to derive the null and preferred directions. Furthermore, the lateral inhibition network could be located extraretinally, such as in the lateral geniculate body or the visual cortex, although this would not account for the observed directional specificity of retinal ganglion cells.

A more careful consideration of Fig 24 suggests that each group of four receptors might provide outputs to more than a single cell. This would make more economical use

of the receptors, and could result in a scheme in which acuity were not degraded. In fact, if a second set of horizontal, bipolar, and ganglion cells were concurrently receiving receptor outputs, but with "reverse" organization, the opposite preferred direction could be derived. The reverse organization is also shown in Fig 24, but smaller and connected by dashed lines. This scheme represents a considerable increase in economy over the previous circuit.

The neural circuit of Fig 24 does have the advantage of an improved nulling in the null direction. The reason for this is that the target hyperpolarizes each bipolar cell before exciting it. With the model of this thesis, an output must occur before inhibition can occur. Thus the null direction can never have a true null. This seems less than tragic when a possible use of such direction specific cells is considered. Presumably such cells come in pairs. That is, for a cell having upward null and downward preferred, there should also be a cell having upward preferred and downward null directions serving the same receptive field. Then, in addition to providing signals to the visual association area of the cerebral cortex, these neurons might be responsible for proper initiation of a reflex such as eye movement for target tracking. Thus, so long as the output from the neuron having its preferred direction in the actual direction of target motion were substantially stronger than the neuron which should be nulled, the proper reflex action should result. Additionally, because visual processing usually occurs in stages, perhaps the nulling could be improved in follow-on processing stages.

By sending the output of the null/preferred direction network to a target neuron with an oscillatory PSP, the result is an overall circuit capable of firing strongly only when a target in a specific direction at a

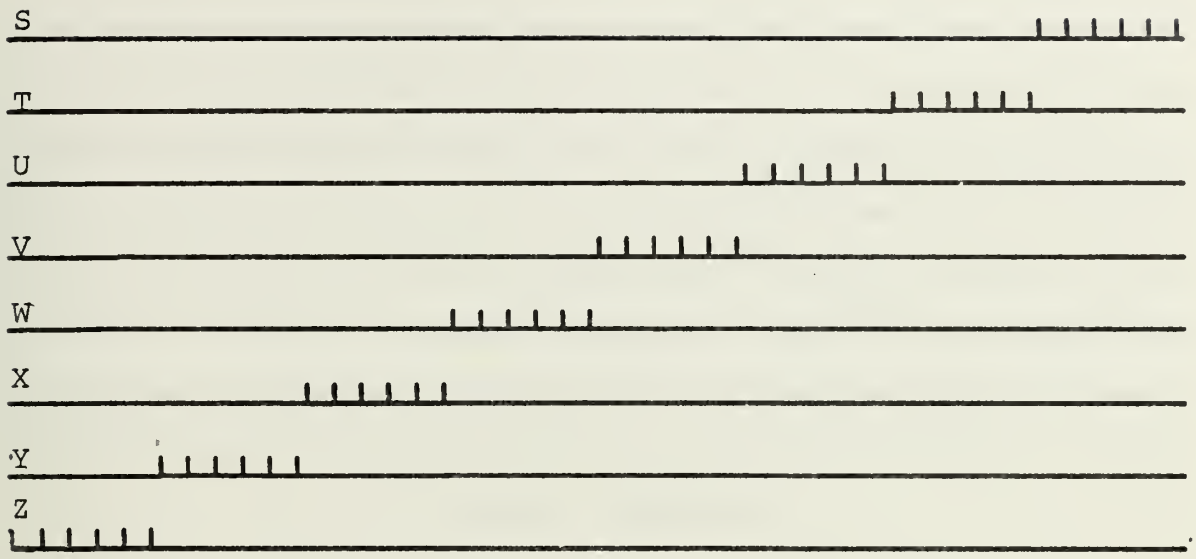
specific speed is present, as might be found in the pons. Such fine grained speed differentiation has not been observed retinally, where only high versus low speed detectors have been proven to exist.

c. Fast Pass

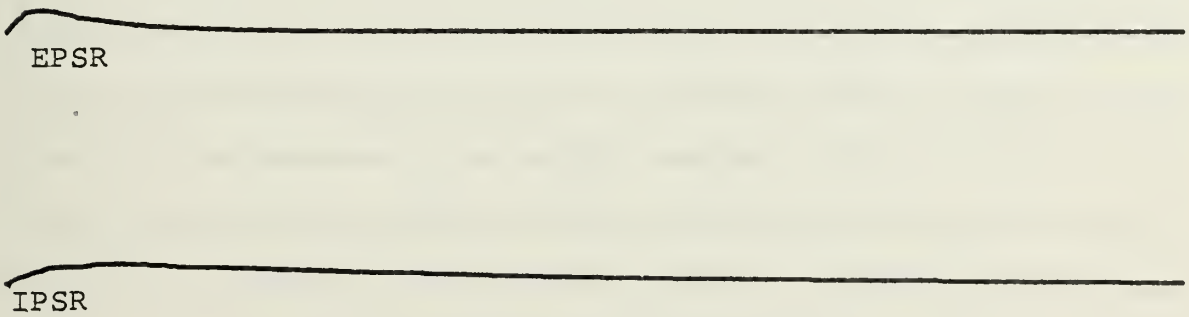
The next model designed was a network of the type found in the retina of the rabbit, which would detect only fast targets. The lateral inhibition network of Fig 6 can be used very nicely to detect only fast targets. Figures 25 and 27a rget in this network. The heart of the high speed pass behavior of this network is the fact that the inhibition of neighboring neurons is delayed in ramp fashion instead of the usual constant delay. This can be seen by observing the spread of inhibition in Figure 27a. A target which is fast enough is able to excite neuron S to produce an output, and then excite neuron T to output before neuron T is inhibited by neuron S. This network has a very abrupt velocity dividing line between detection and no detection.

A slow target illuminating the high pass network is depicted in Figs 26 and 27b. Here, the inhibition resulting from the output of neuron S spreads to neurons T, U, V, W, and X in time to inhibit them before the arrival of the target illumination. Neuron Y is beyond the influence of inhibition by neuron S, and therefore has an output. This network could be made more effective, then, by altering the LID table such that inhibition would spread farther.

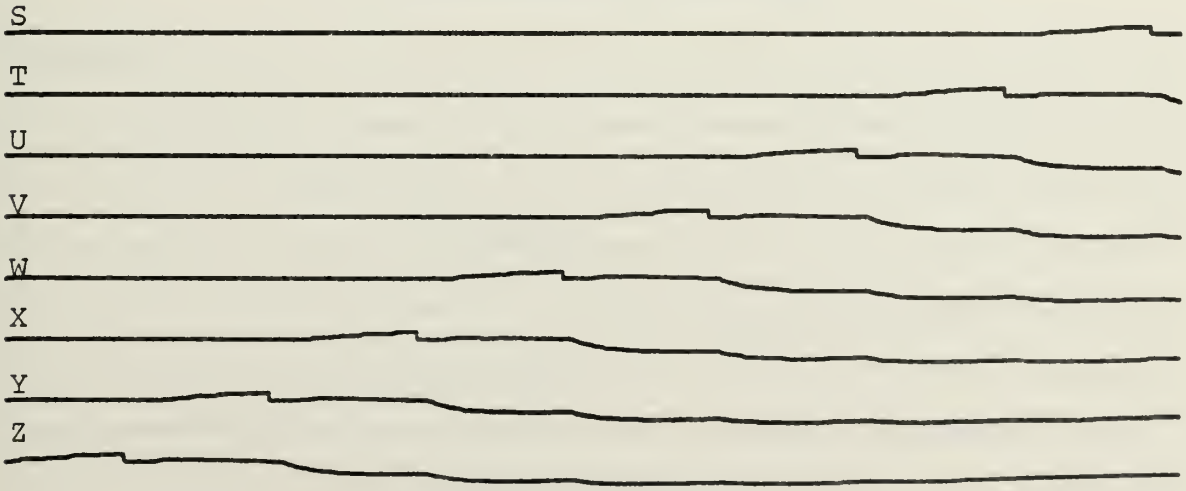
In Fig 27, note that the ramp delay in spread of inhibition consists of incrementel delays of about ten milliseconds. This could be accomplished by several synaptic delays [Ref 8].



A. INPUT, PREFERRED

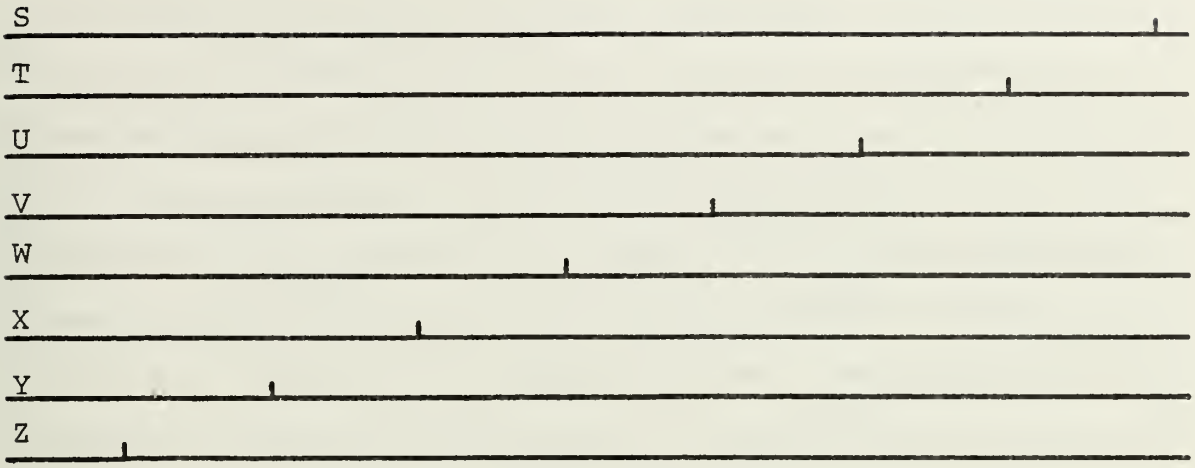


B. PSR, PREFERRED AND NULL

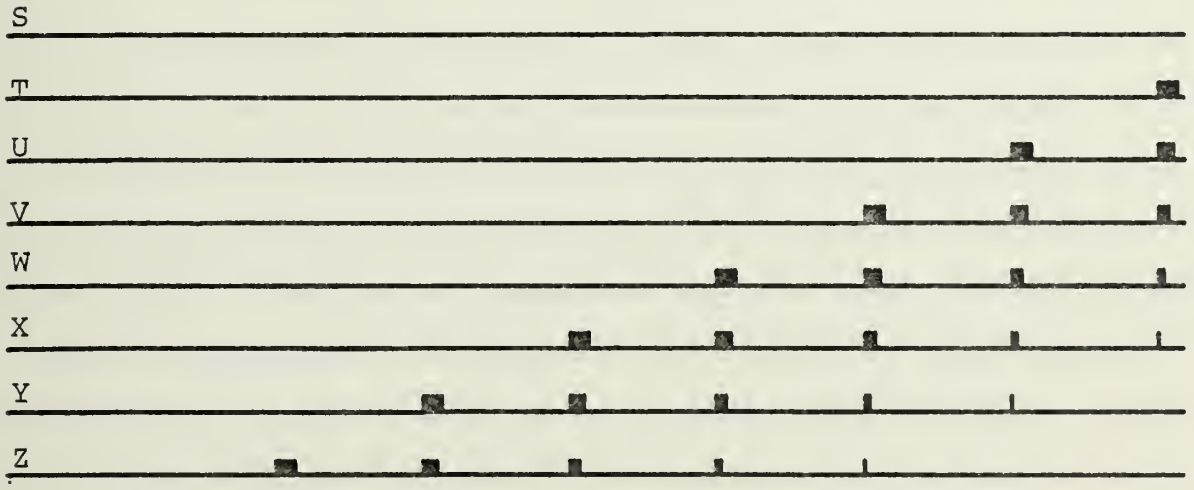


C. PSP, PREFERRED

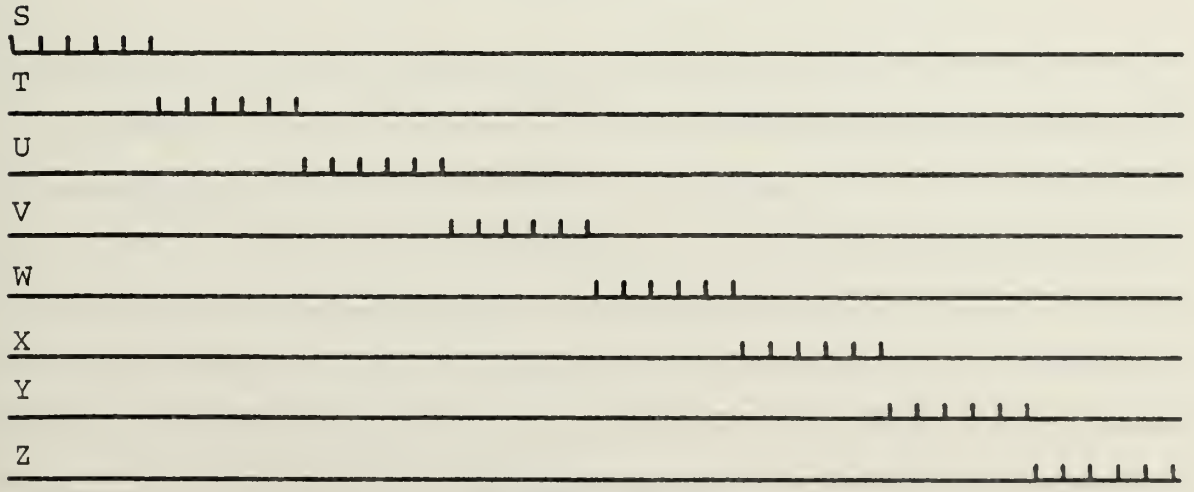
FIGURE 21 - NULL VERSUS PREFERRED DIRECTION, PART 1



A. OUTPUT, PREFERRED

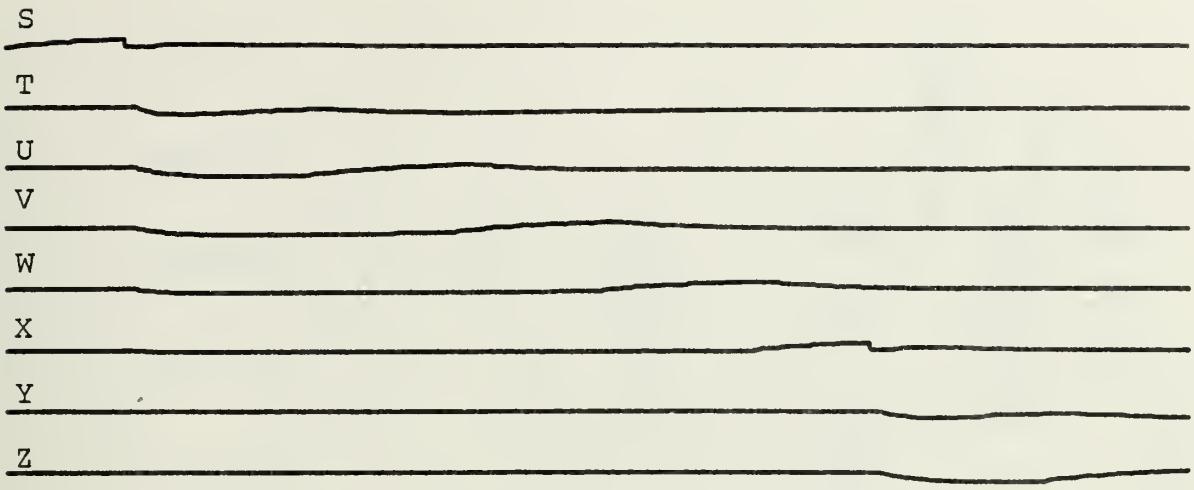


B. INHIBITION, PREFERRED

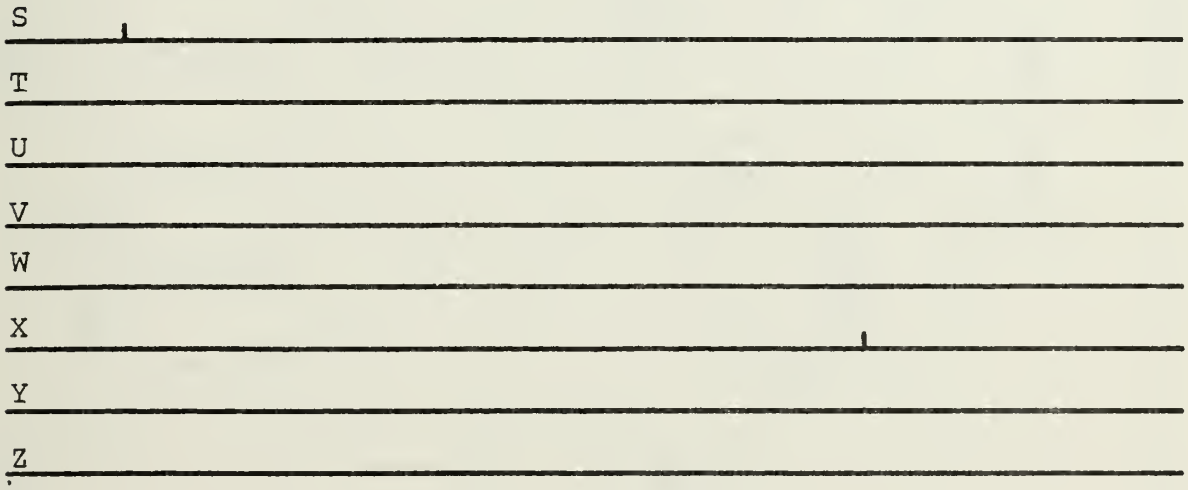


C. INPUT, NULL

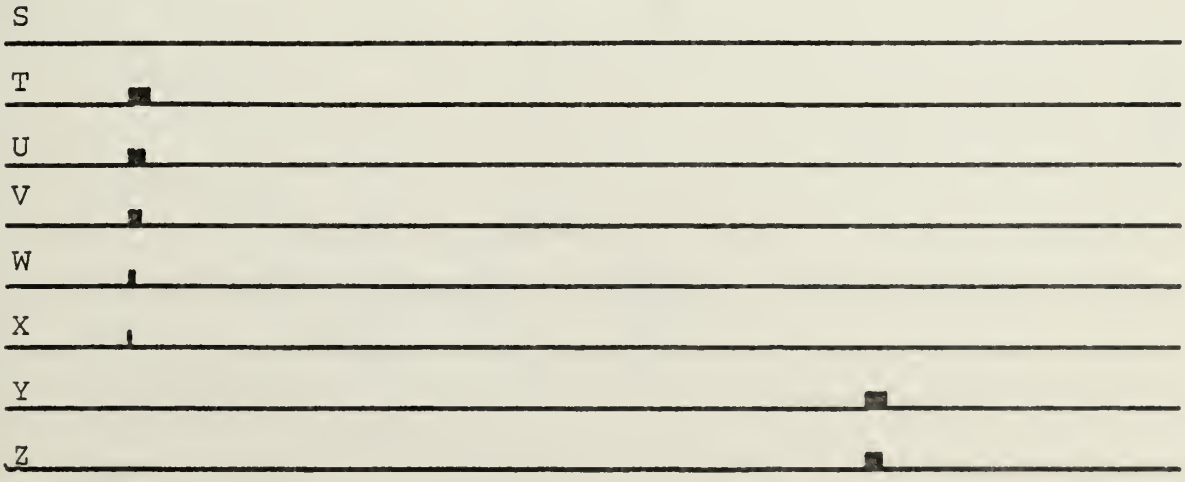
FIGURE 22 - NULL VERSUS PREFERRED DIRECTION, PART 2



A. PSP, NULL



B. OUTPUT, NULL



C. INHIBITION, NULL

FIGURE 23 - NULL VERSUS PREFERRED DIRECTION, PART 3

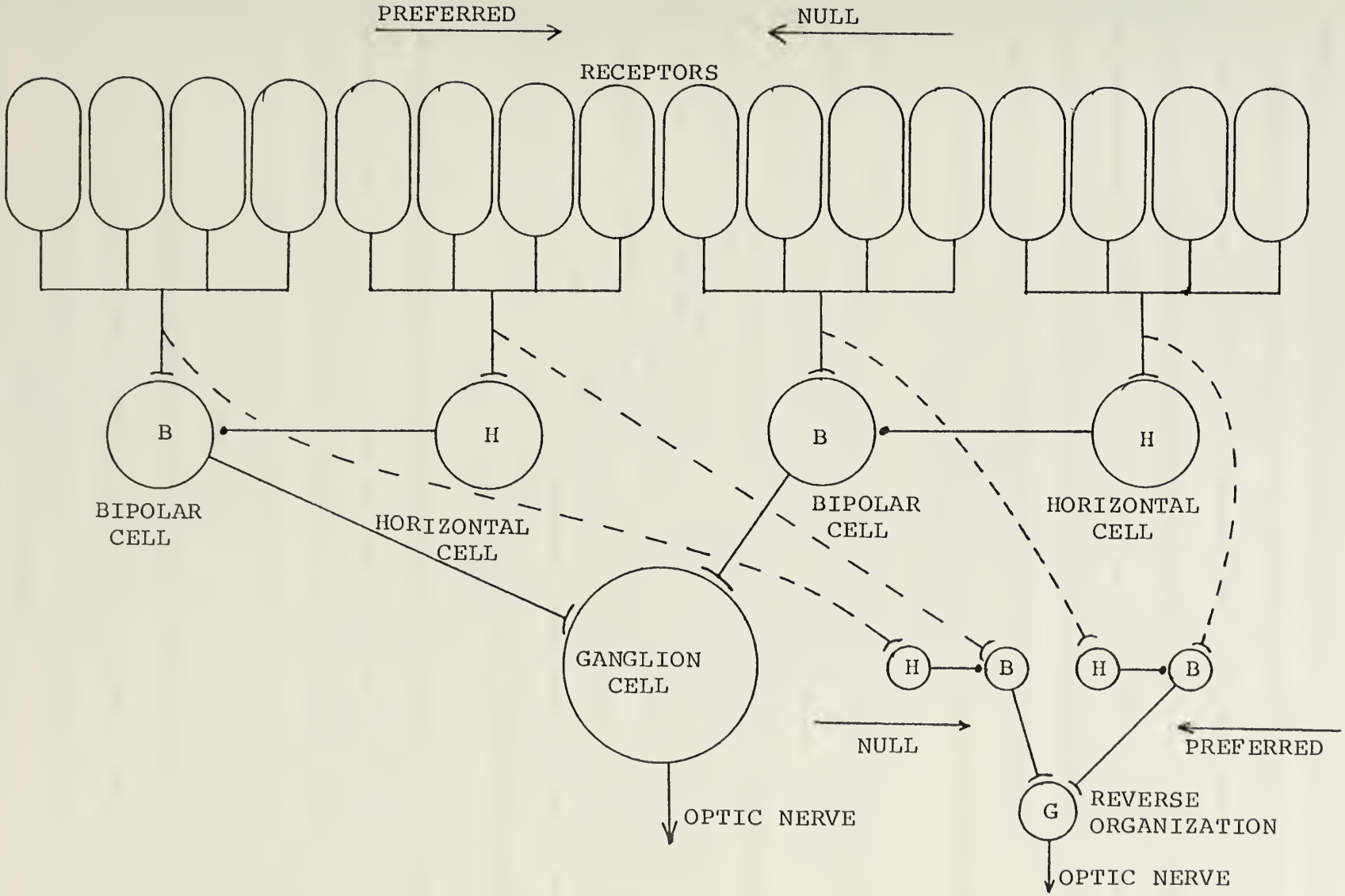
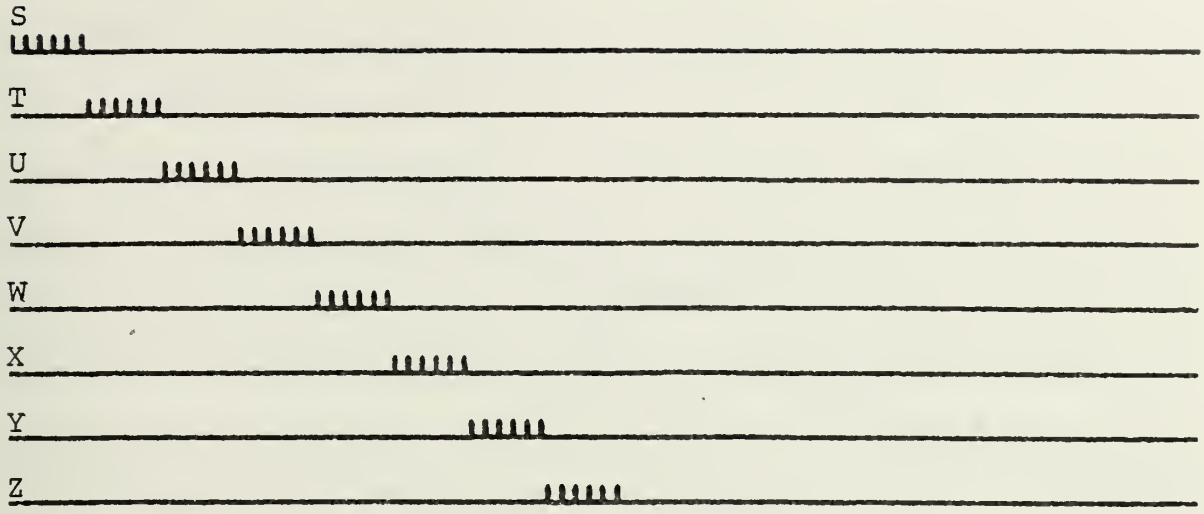
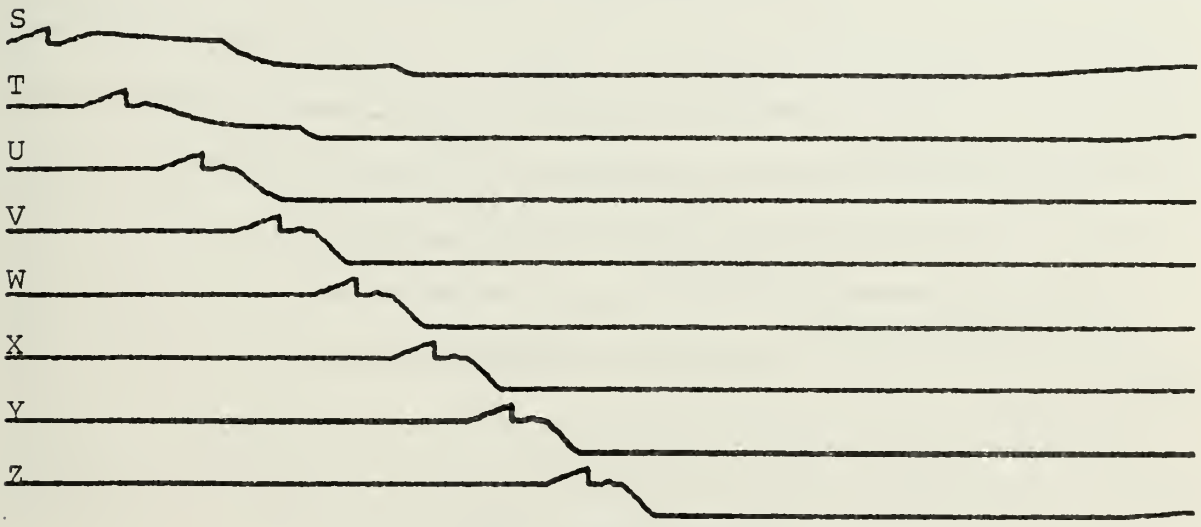


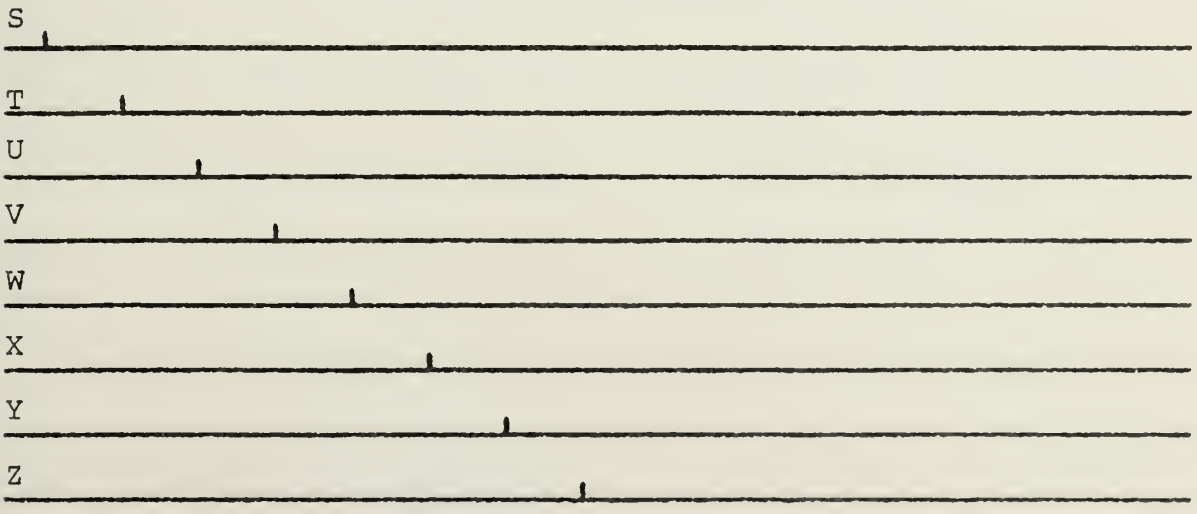
FIGURE 24 - CIRCUIT FOR NULL VS. PREFERRED DIRECTION



A. INPUT, FAST TARGET

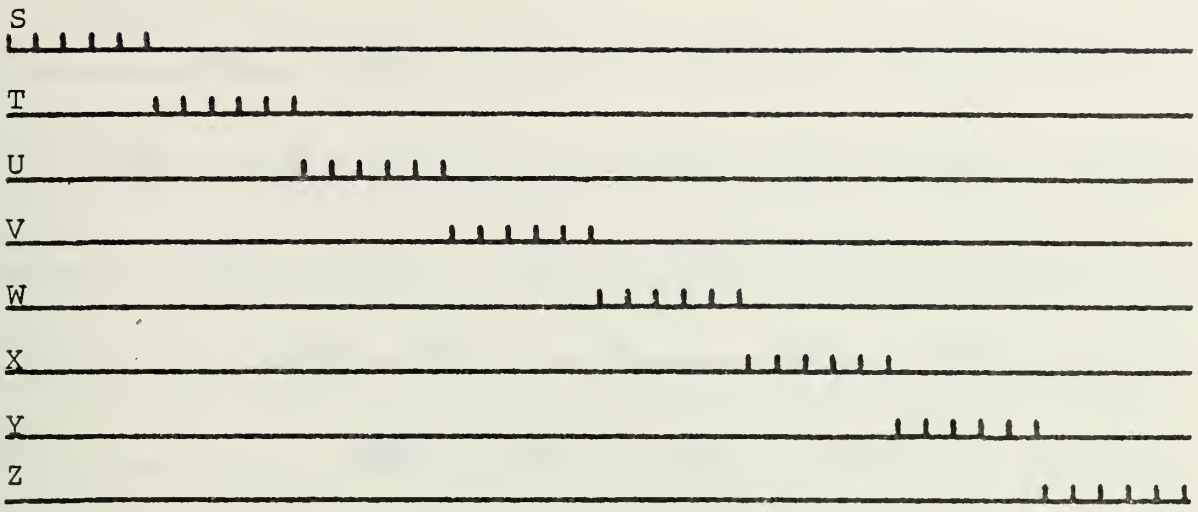


B. PSP, FAST TARGET

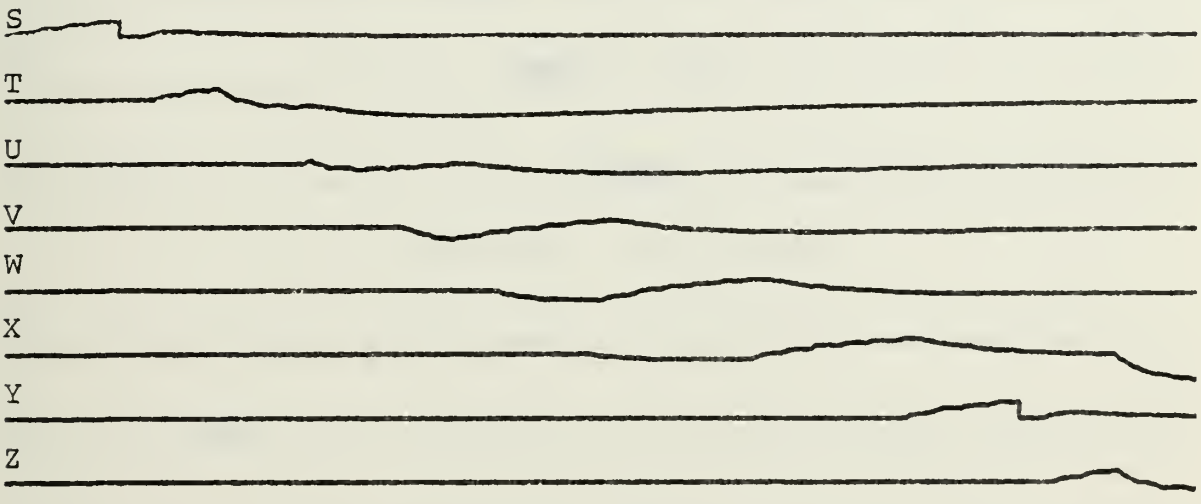


C. OUTPUT, FAST TARGET

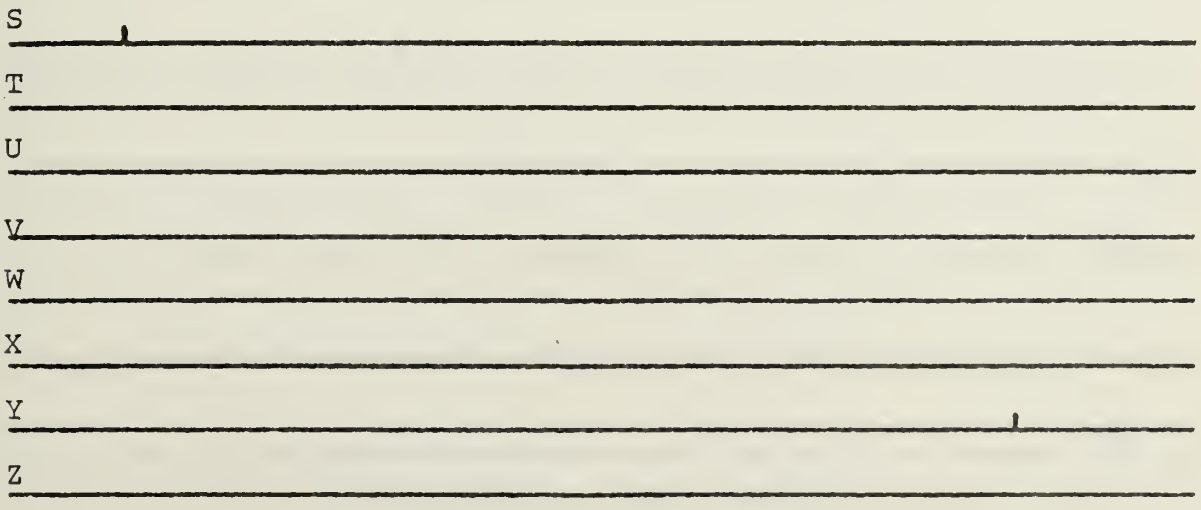
FIGURE 25 - FAST PASS DETECTOR, PART 1



A. INPUT, SLOW TARGET

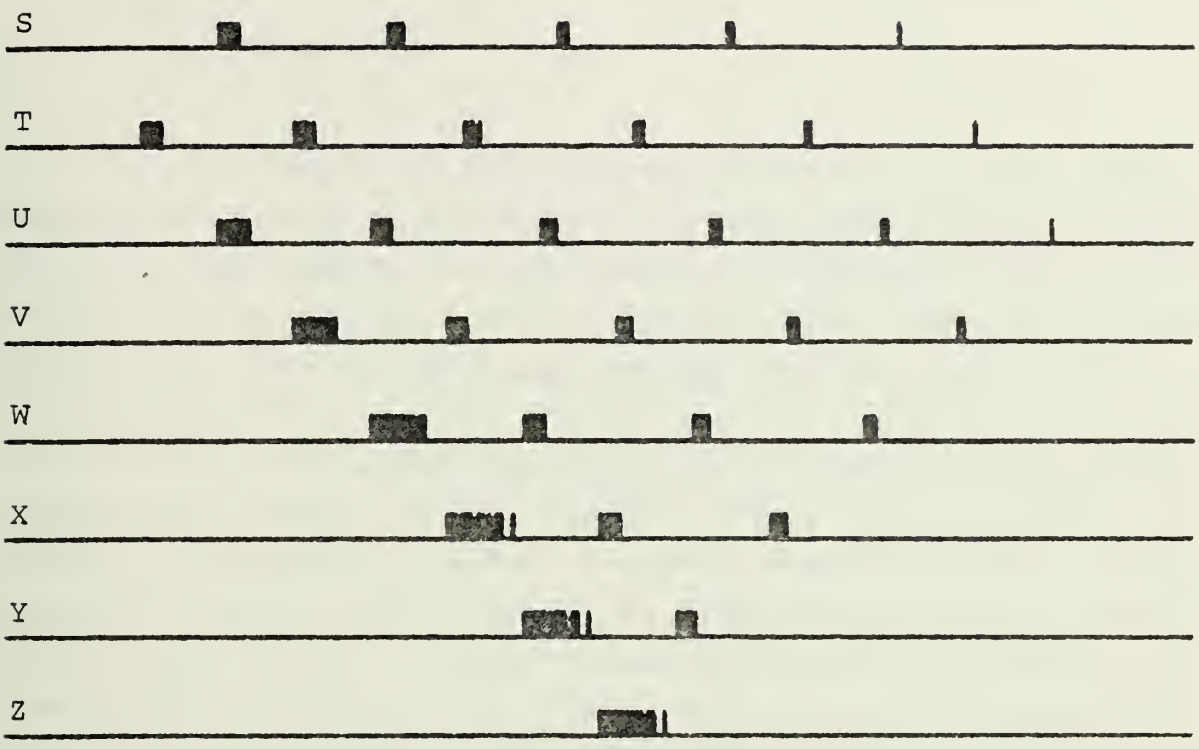


B. PSP, SLOW TARGET

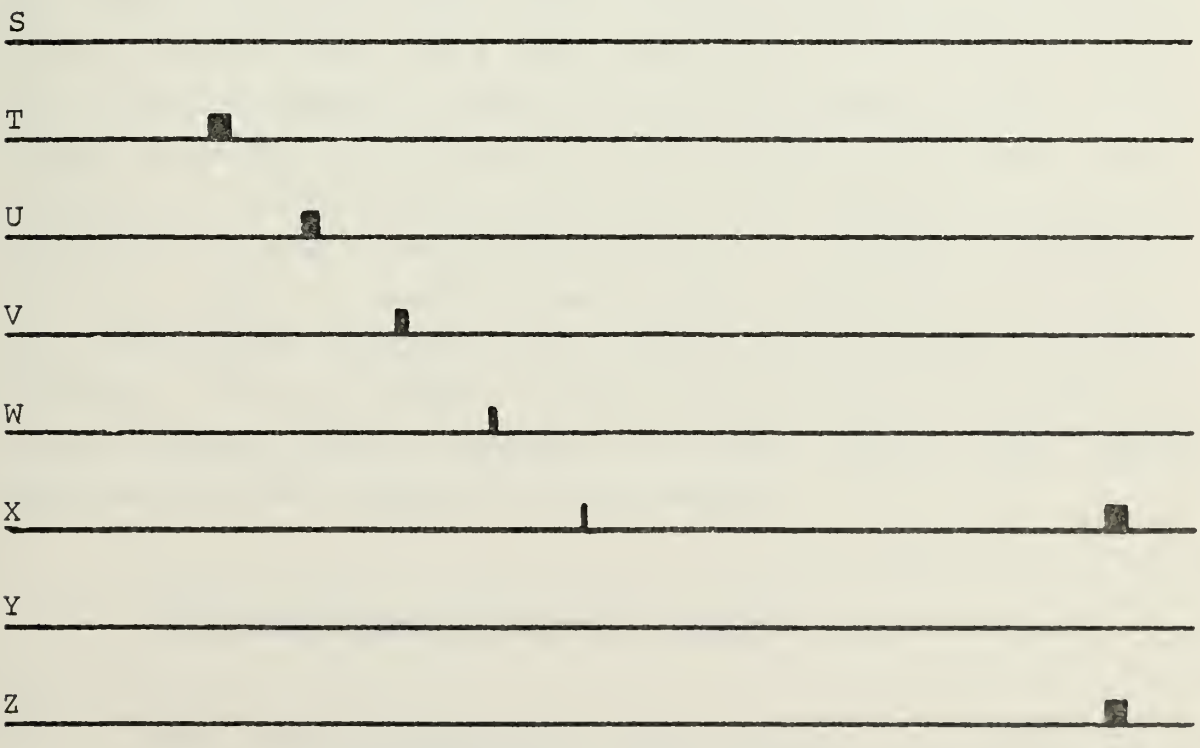


C. OUTPUT, SLOW TARGET

FIGURE 26 - FAST PASS DETECTOR, PART 2



A. INHIBITION, FAST TARGET



B. INHIBITION, SLOW TARGET

FIGURE 27 - FAST PASS DETECTOR, PART 3

d. Slow Pass

LINHIB can also be used to model a network which detects slow targets but fails to detect fast targets. This is done by using a constant delay, or perhaps a small ramp delay, and by carefully selecting the IPSR duration. The plots for such a slow pass network for both slow and fast targets are shown in Figs 28, 29, and 30. First, for the slow target, the inhibitory influence on neuron T due to the outputs from neuron S are largely gone by the time the target illuminates neuron T, and an output results. Only neuron Z fails to fire, but it is probable that this neuron would fire given several more milliseconds, judging from comparisons of neuron Z's PSP with that of neuron Y. Therefore, let us say that all eight neurons fired or would have fired. A high speed target is now used to illuminate the same network (Fig 29). Note that here, only five of eight neurons fired for a speed ratio of two to one. This may seem a rather modest result, but consider that in a single pole filter, a frequency ratio of two to one would produce an attenuation of only six decibels, which is comparable to the five to eight ratio. A subtle additional fact is that a double speed target would produce roughly half the number of inputs per channel to the network. Taking "credit" for this result of high speed, the characteristics of the network improve somewhat (Fig 30). Here only three neurons fire, as opposed to five before.

5. Regularization of Random Inputs

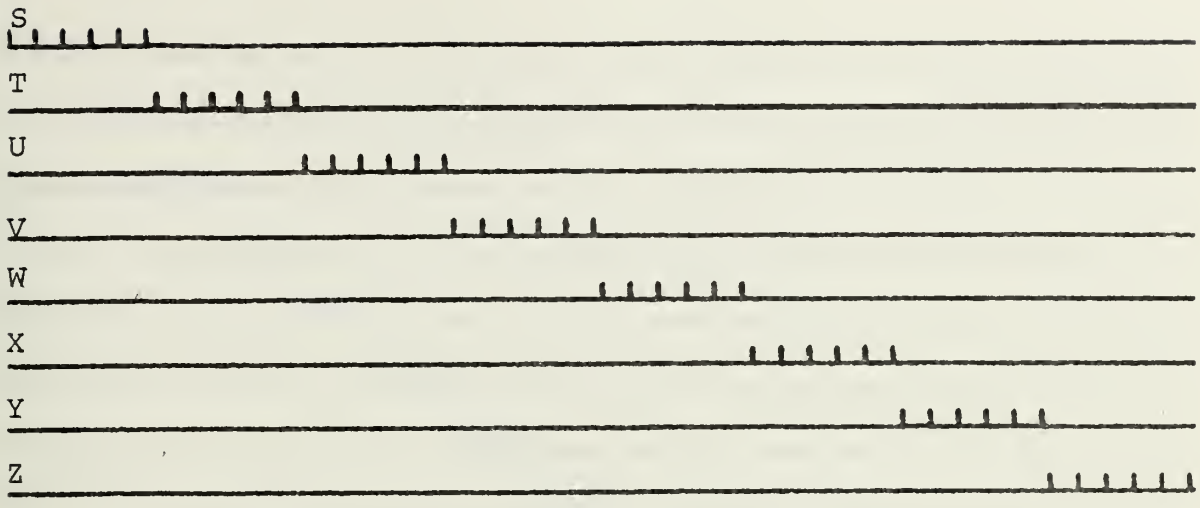
The normal approach to modeling is to determine what hard information exists about the system to be modeled, build the model, and then use it to determine its usefulness

in duplicating the behavior of the real system. Once confidence in the model has escalated somewhat, the modeler can begin to extend application to areas not necessarily observed in real life. Then if an interesting output results from the model, two possibilities exist. First, the model might really not be an adequate description of the real system, and the ineresting result worthless. Second, the model could be valid, and the result could be worth looking for in the real system.

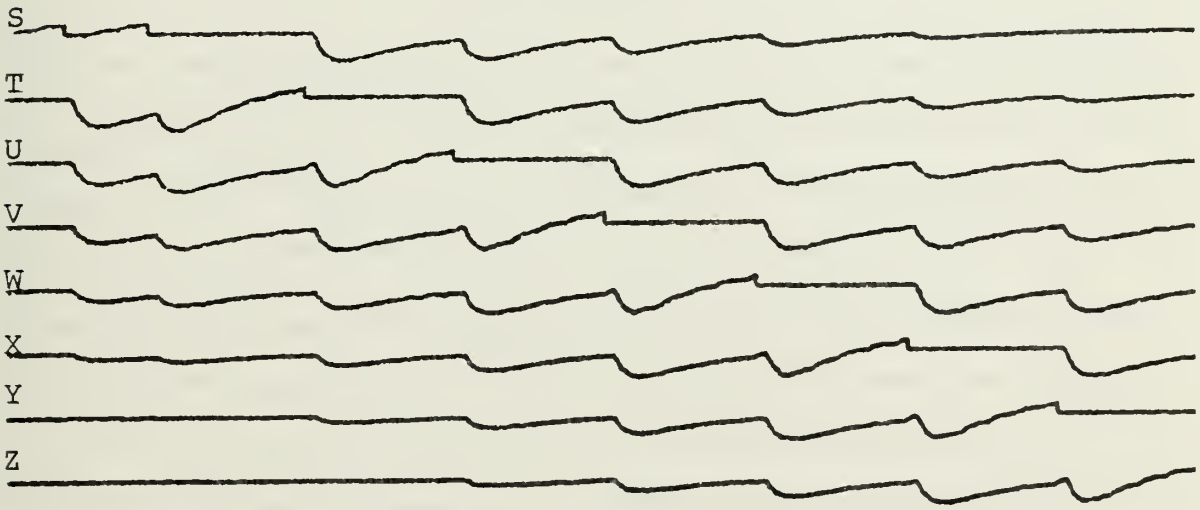
One such result observed while using the lateral inhibition network with random inputs is that an unrandomized output can be produced (Figs 31 and 32). The summed output plot, while not completely regular, is nonetheless much more regular than any of the inputs. Such orderliness results from the very character of the lateral inhibition network. Namely, all channels are vying for output production. When an output occurs, all other nearby channels are inhibited for the duration of the IPSR. This means that during this interval, nearby outputs are unlikely. Since this interval would tend to be the same after each output, the result is a rather uniform time between outputs. More complete regularization would occur if the inhibitory influence were to spread farther. In support of this notion, note that the two closest outputs (the third and fourth) in the summed output plot originated from spatially distant channels. If inhibitory spread had been more complete, then in all likelihood, the fourth output would not have occurred so soon.

This method of regularizing random inputs is to be contrasted with that of Fig 17, where an oscillatory PSP was needed.

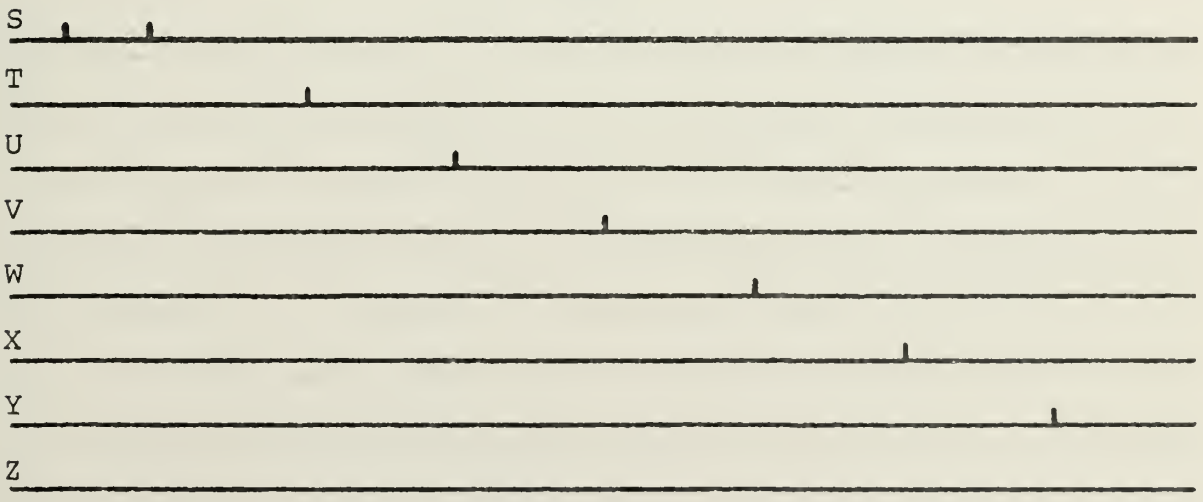
There are possible uses for an orderly, regularly spaced spike train. In the next section it will be shown



A. INPUT



B. PSP



C. OUTPUT

FIGURE 28 - SLOW PASS DETECTOR, SLOW TARGET

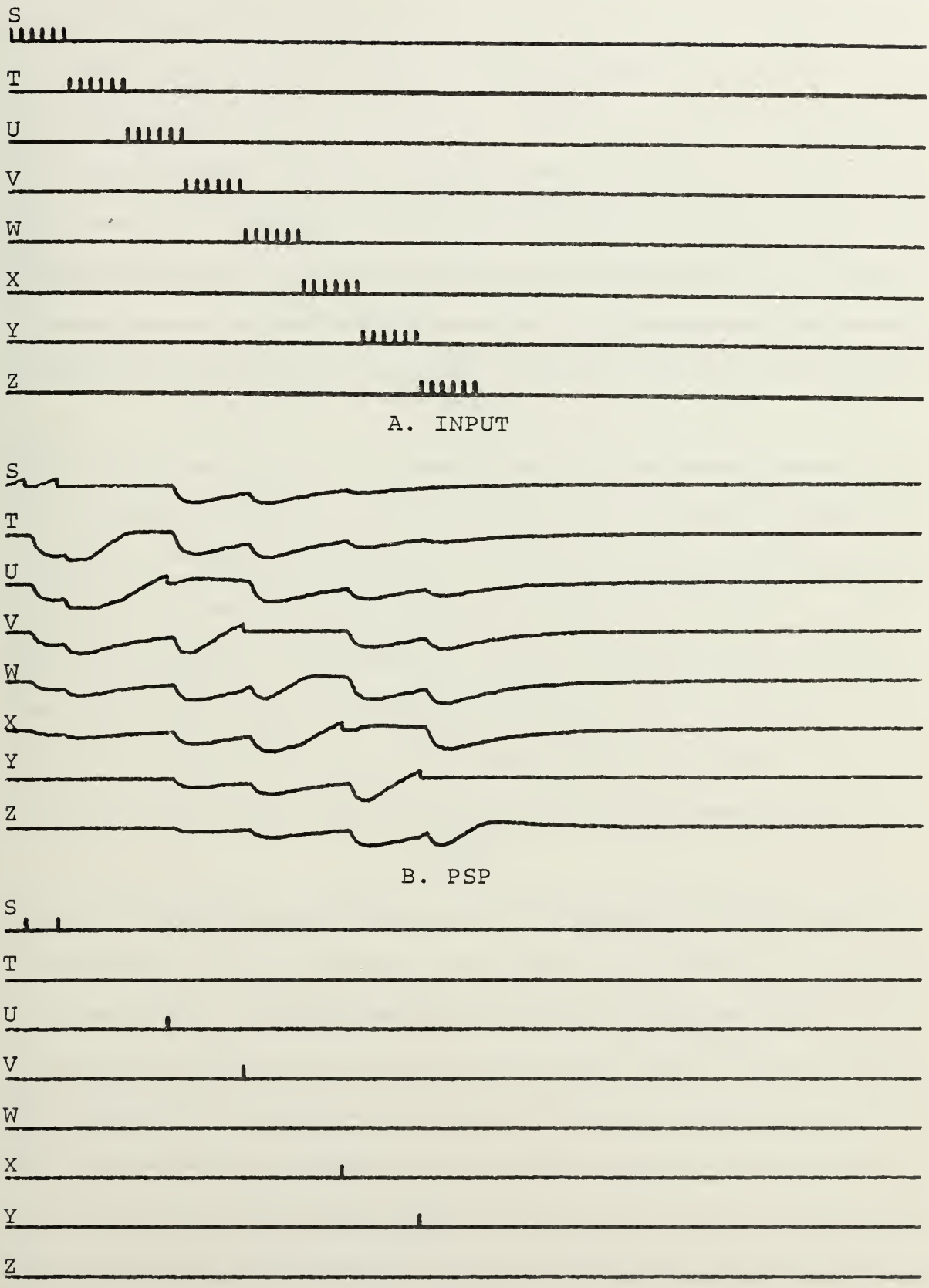
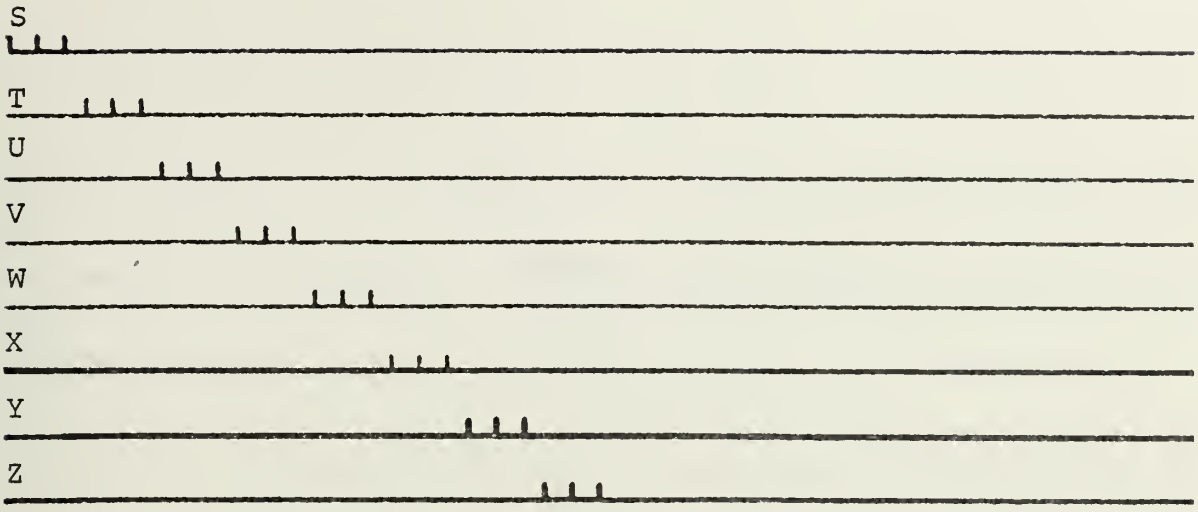
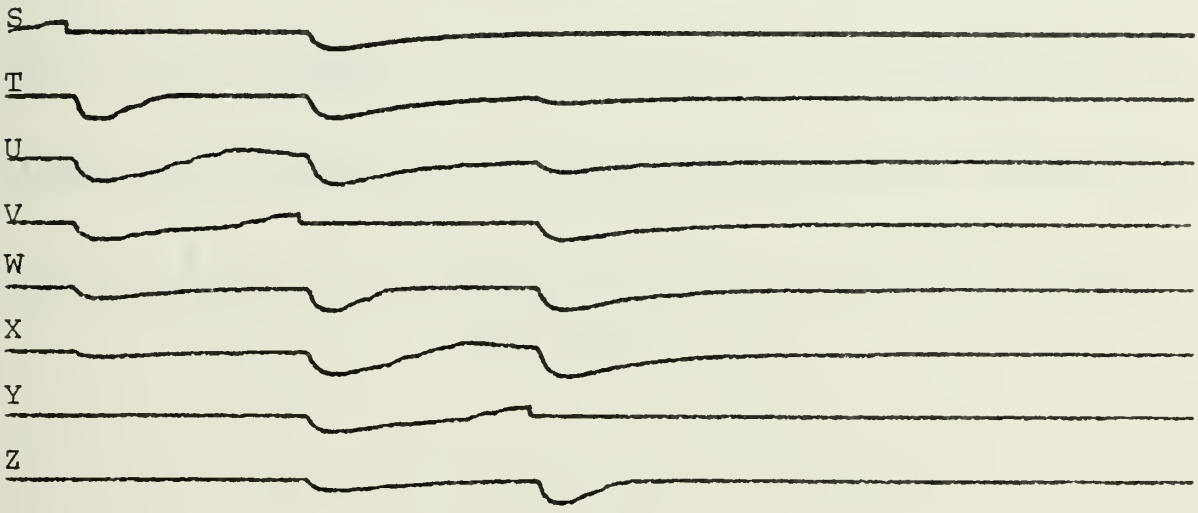


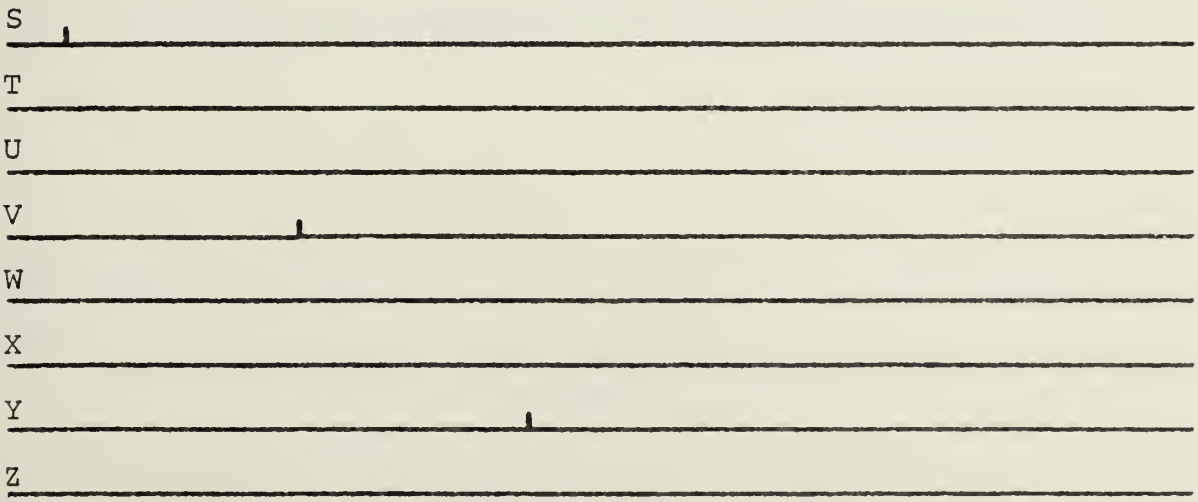
FIGURE 29 - SLOW PASS DETECTOR, BRIGHT FAST TARGET



A. INPUT

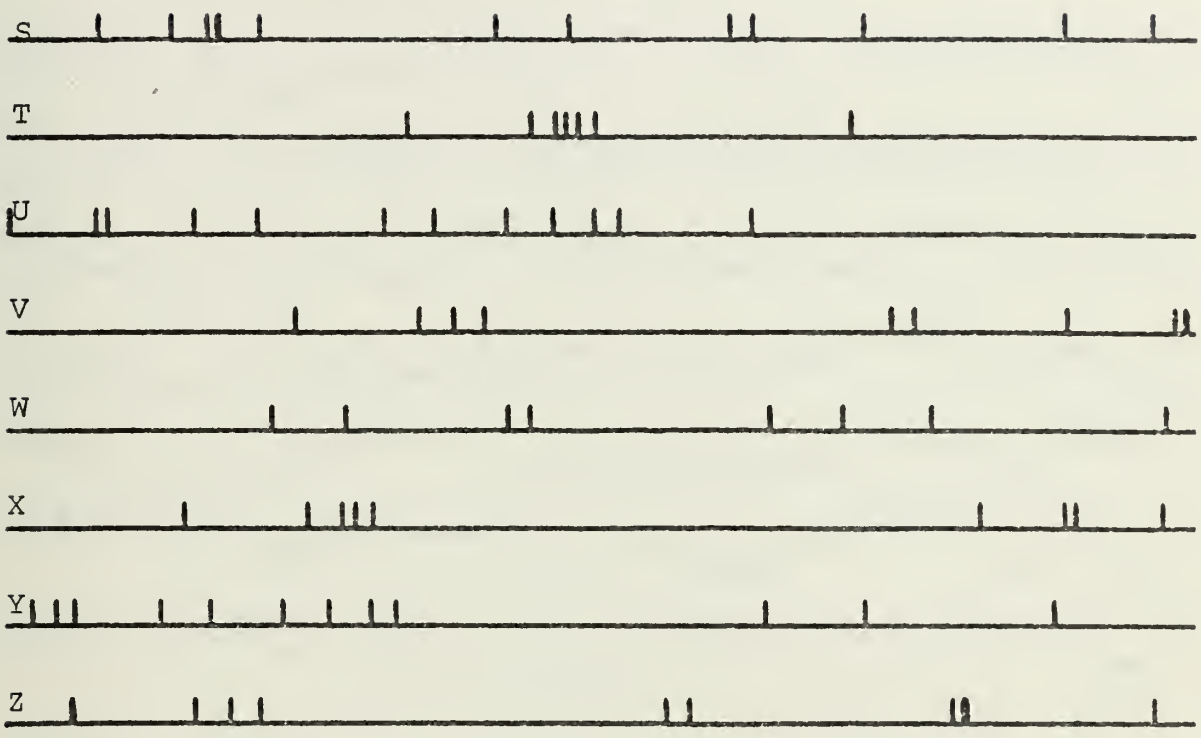


B. PSP

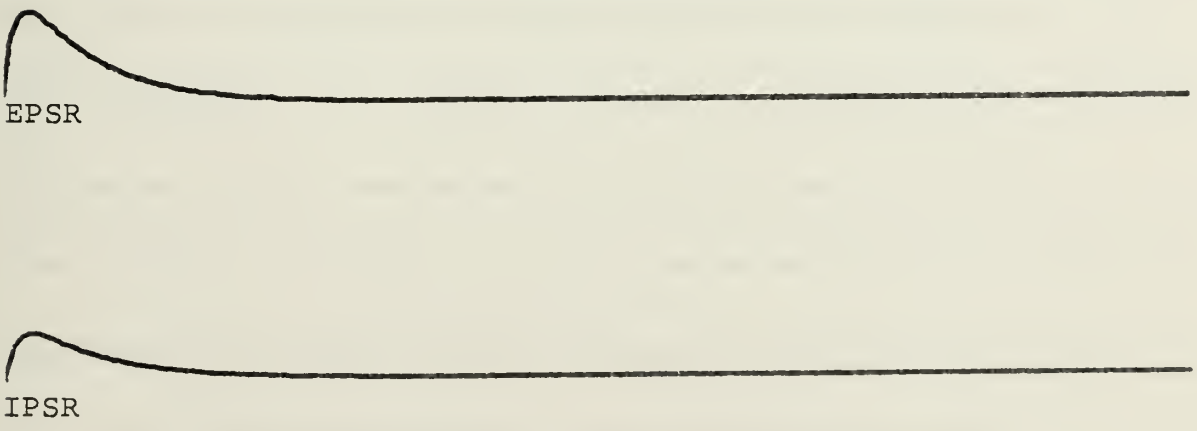


C. OUTPUT

FIGURE 30 - SLOW PASS DETECTOR, NORMAL FAST TARGET

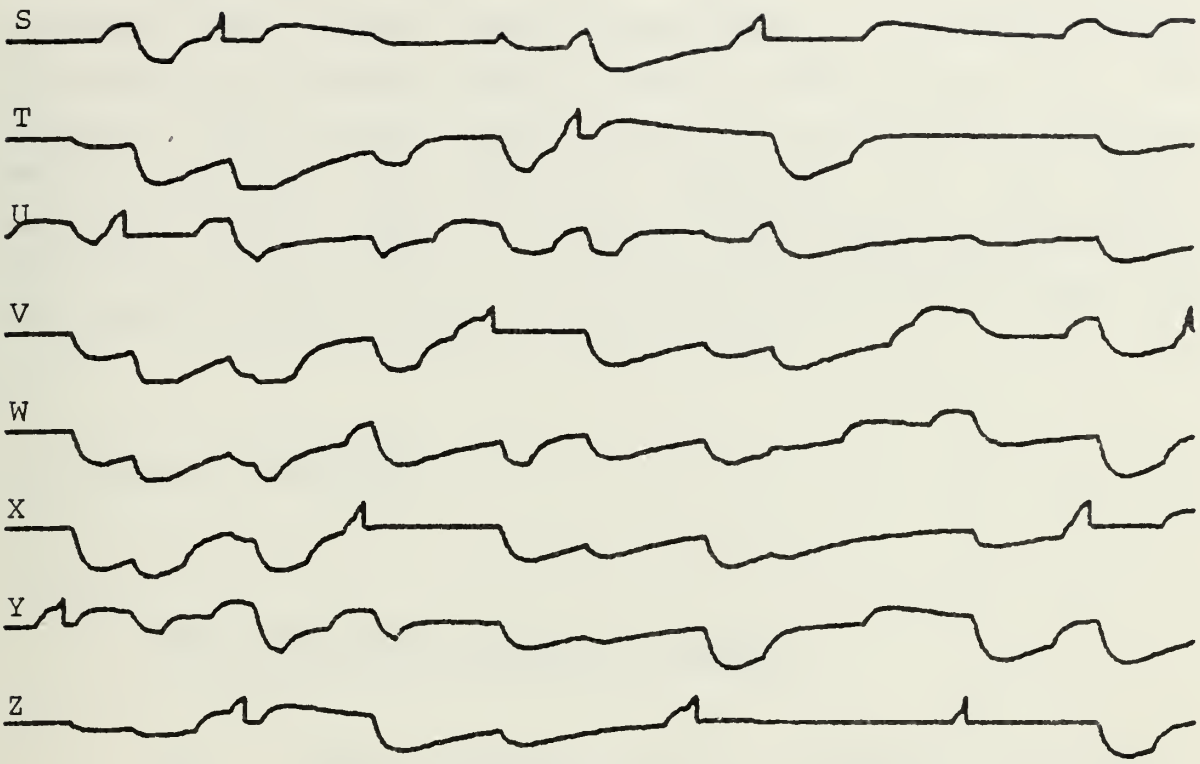


A. INPUT

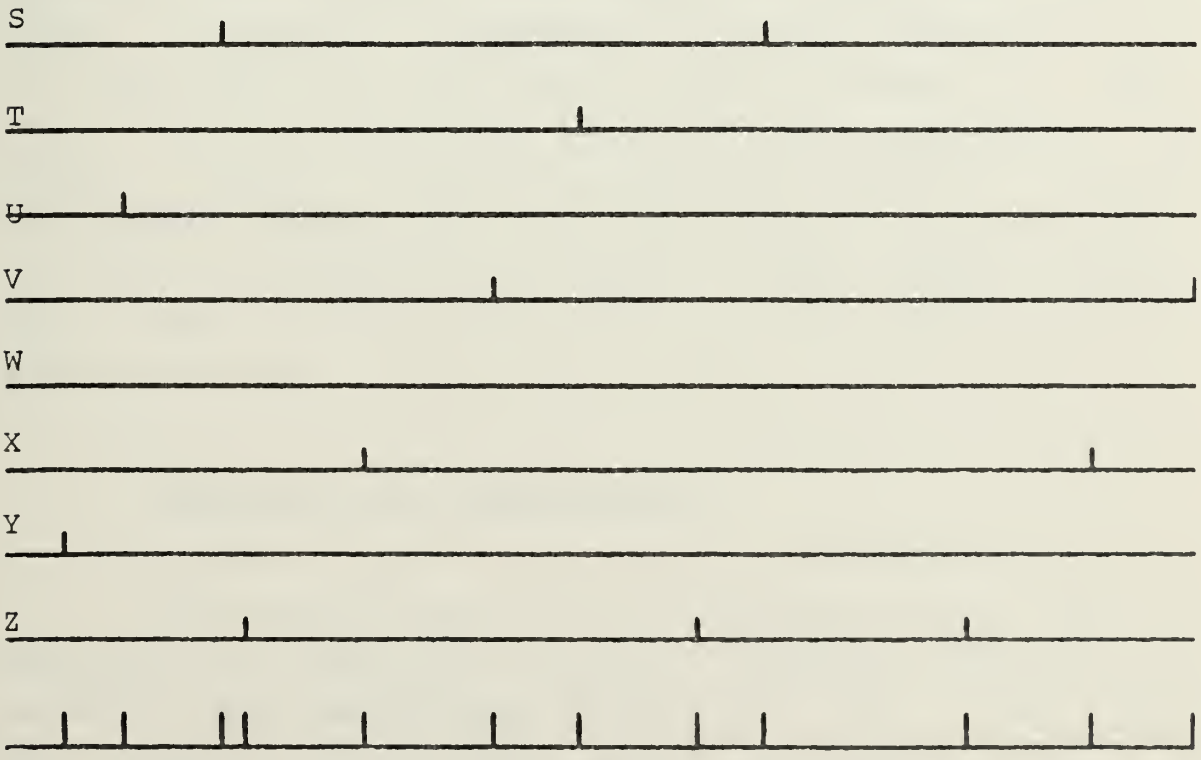


B. PSR

FIGURE 31 - REGULARIZATION BY LINHIB, PART 1



A. PSP



B. OUTPUT

FIGURE 32 - REGULARIZATION BY LINHIB, PART 2

that a regularly spaced set of inputs to an inhibitory network has extreme phase sensitivity, so long as the channels have roughly equal firing rates. That is, the channel whose input phase allows it to produce the first output will gain and maintain dominance over the other channels. This could be considered as a possible mechanism for a person being able to direct his attention to only one of several sensory inputs of equal level.

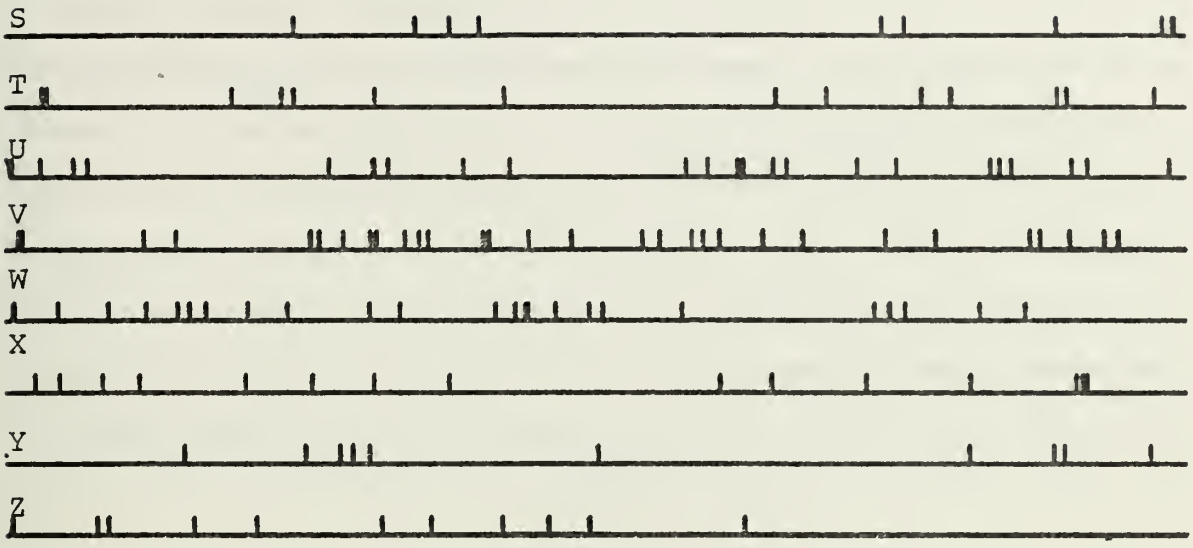
C. SPATIAL PHENOMENA

1. Basic Spatial Behavior of LINHIB

As a basic example of the operation of LINHIB, consider Figs 33 and 34. The inputs are random, but are most dense in channel V, decreasing in either direction. It is not surprizing that more outputs occur in channel V, since more inputs were provided there. But more importantly, the ratio of outputs to inputs is highest for channel V, because of lateral inhibition. That is, channel V gains dominance over the other channels by having outputs which inhibit them. Thus it is more difficult for the other channels to produce outputs.

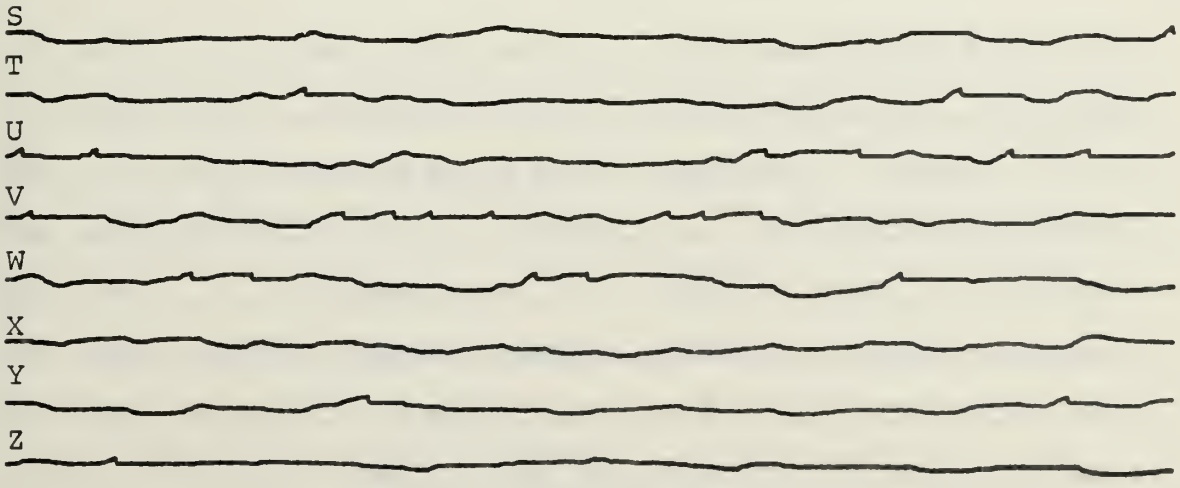
2. Inhibition and Disinhibition

The two most basic spatial phenomena which can be demonstrated using the lateral inhibition network are inhibition and disinhibition (Figs 35 and 36). Note that only three of the eight neurons have inputs, and that at first, only two have inputs. Neuron X gains dominance over Neuron Z by producing an output first. Because



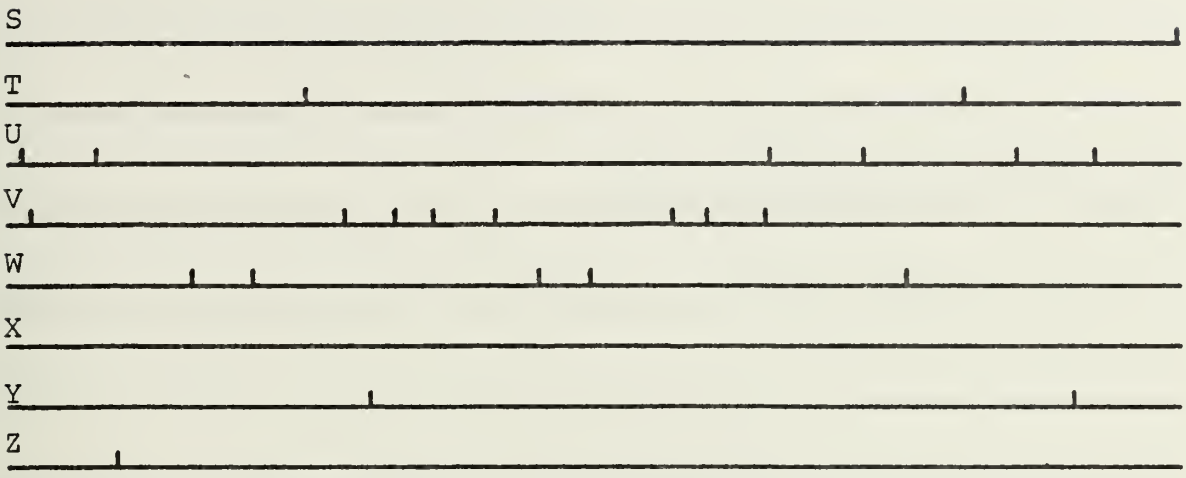
A. INPUT

B. PSR



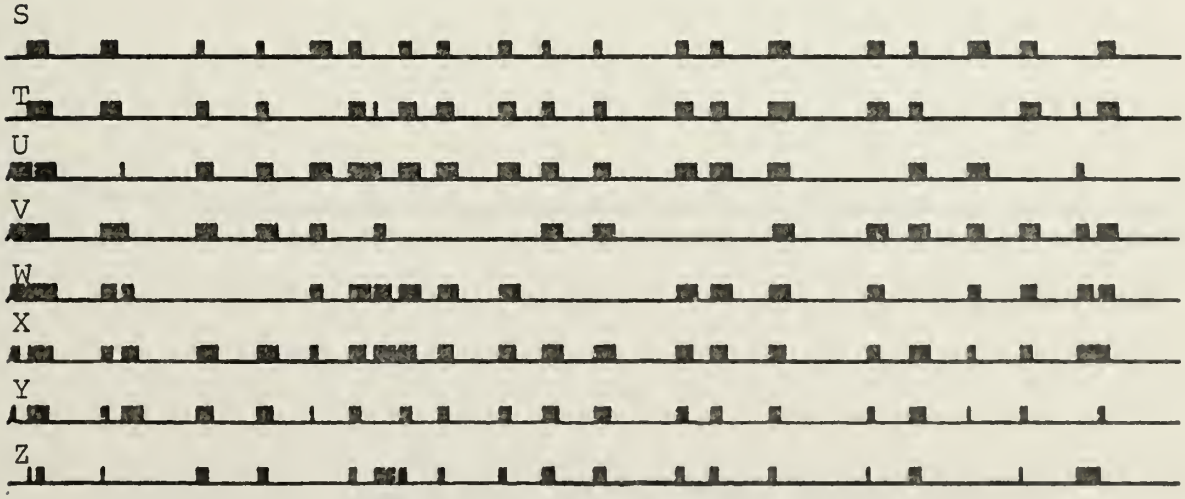
C. PSP

FIGURE 33 - BASIC SPATIAL BEHAVIOR OF LINHIB, PART 1



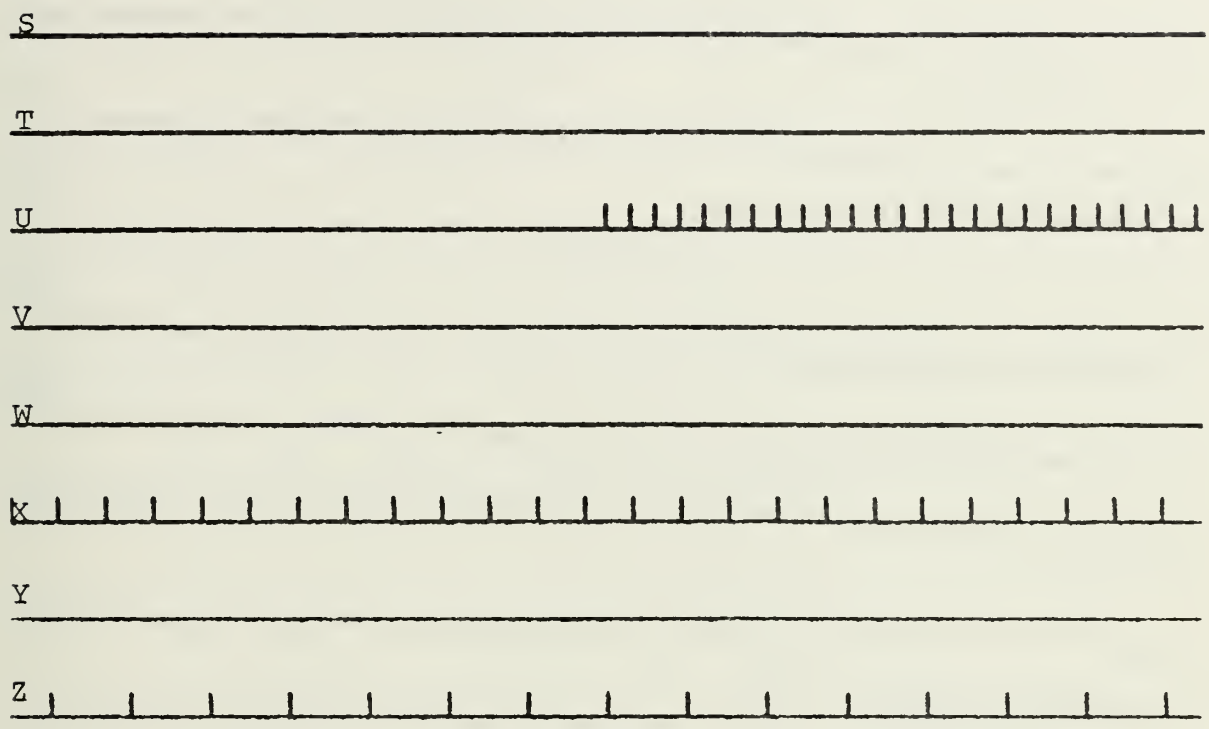
A. OUTPUT

B. SUMMED OUTPUT



C. INHIBITION

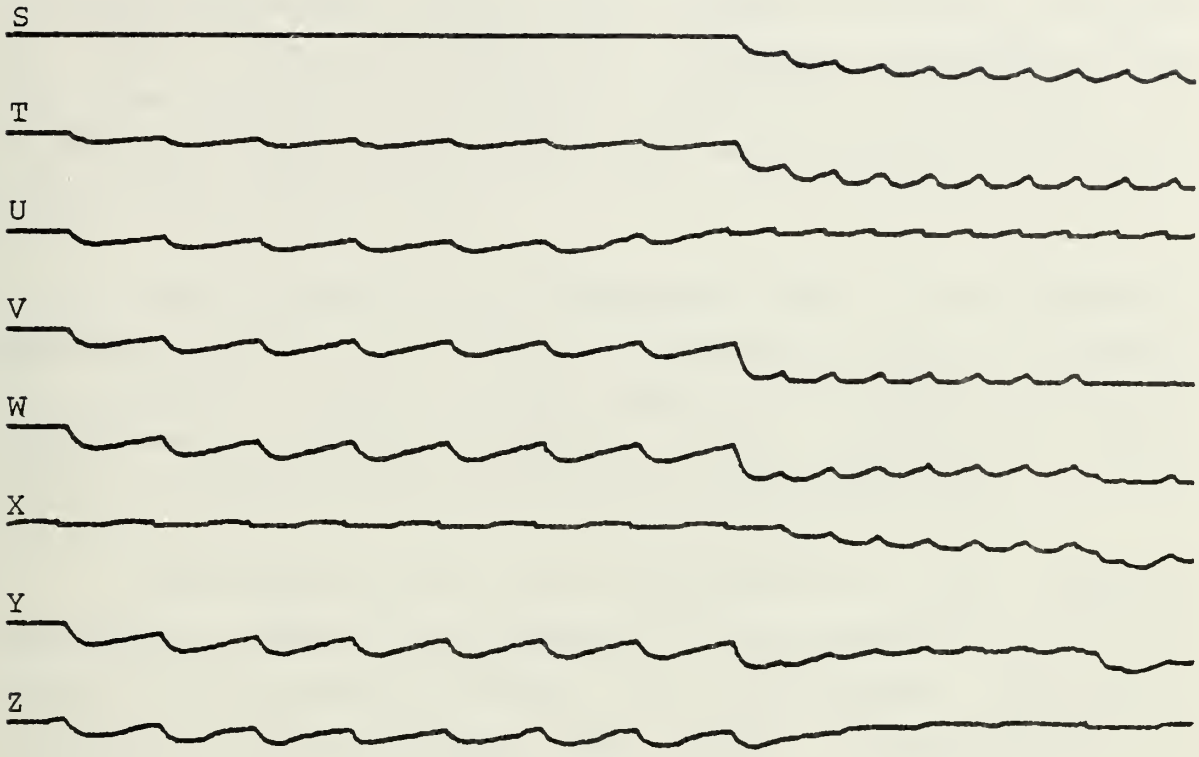
FIGURE 34 - BASIC SPATIAL BEHAVIOR OF LINHIB, PART 2



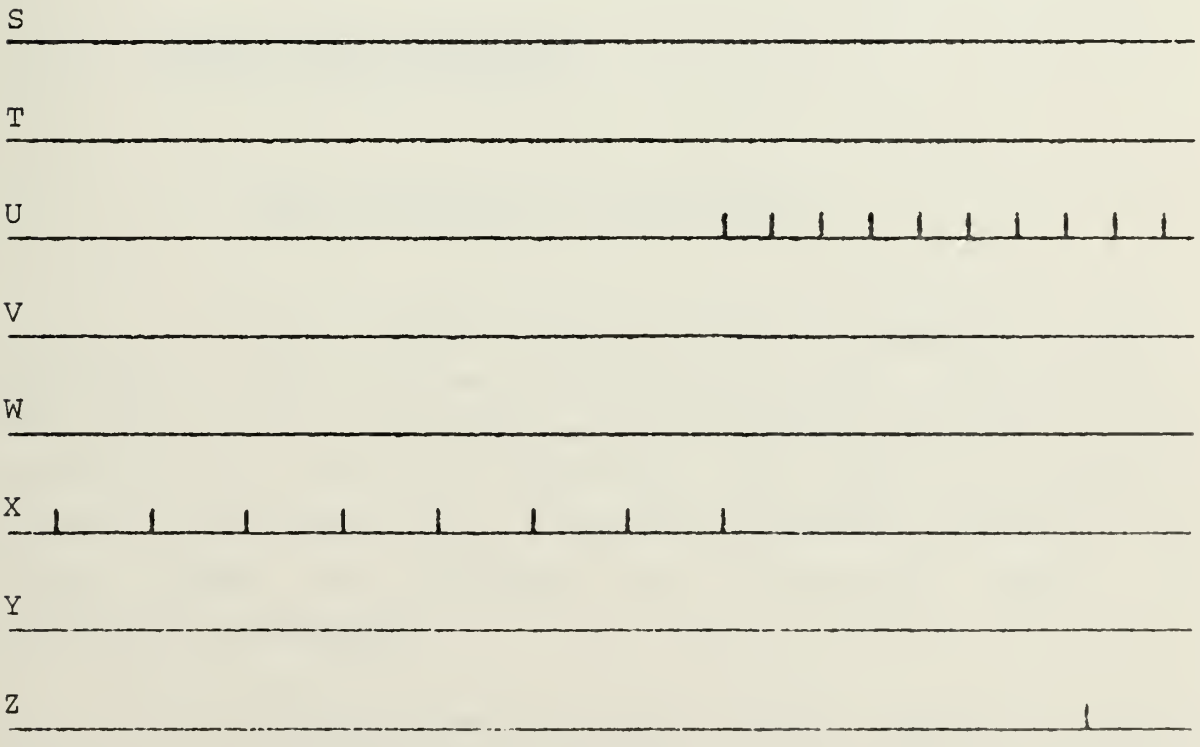
A. INPUT

B. PSR

FIGURE 35 - INHIBITION AND DISINHIBITION, PART 1



A. PSP



B. OUTPUT

FIGURE 36 - INHIBITION AND DISINHIBITION, PART 2

LID=000234505432000, neuron Z is strongly inhibited and does not fire, allowing neuron X to maintain dominance. This describes inhibition of neuron Z, and would be analagous to a microelectrode measurement of neuron Z while providing a bright surround stimulus.

When neuron U is illuminated with an even brighter stimulus than that for neuron X, neuron U gains dominance and inhibits neuron X. This results in a complete lack of inhibition tc neuron Z, which now fires. (Inhibition from neuron U spreads only as far downward as neuron Y).

Disinhibition has been demonstrated in the limulus eye [Ref 6], but is not important to the organism in its own right. It is, rather, a result of the lateral inhibition network's characteristics, and served to demonstrate the spatial extent of inhibitory spread.

3. Bright Line Phenomena

a. Line Sharpening by Inhibition

One function which a lateral inhibition network performs in vision is the sharpening of lines which become blurred due to scattering in the eyeball's structures. The optics of the eye are excellent, but are not perfect. Lateral inhibition is a viable method for improving the visual system by increasing its resolving power by performing sharpening.

A single, narrow, bright line against a dark background was chosen. A lateral inhibition network will also perfcrm sharpening on a narrow dark line on a bright background, probably just as effectively as the former case.

But since the eight neuron linear array is tacitly flanked by many other neurons in total darkness (no inhibition is fed into the network from above neuron S or below neuron Z), the central bright line was the most reasonable choice.

An important point is that line sharpening occurs whether or not optical blurring occurs. In other words, it is only the photon distribution on the retinal receptors and the exact nature of the neural connections which determine the output to the brain. Neither the retina nor the brain has any way of knowing whether a blurred line distribution on the retina is an optically blurred image of an actual sharp line, or a perfect image of an actual blurred line. That is, a lateral inhibitory network will sharpen <u>any</u> line, whether it is actually sharp or not.

Line sharpening is illustrated in Fig 37. For this run, the strength of one unit of inhibition was chosen to be 1.25 times the strength of one unit of excitation (I=1.25E). This was done by controlling the size of the IPSR. Note that neuron V has the highest intensity input, and is therefore the retinal image of the line's true location. But due to scattering, all channels have inputs to a lesser extent. The outputs of Fig 37c demonstrate that remarkable sharpening has taken place in that only neuron V has outputs. The reason this happens is that neuron V produces the first output and therefore inhibits all other neurons from producing outputs. This and the higher input rate allow neuron V to maintain dominance.

Because of the complete nature of this example of line sharpening, it was decided to lower the IPSR to gain some insight into how much inhibition is needed to give satisfactory sharpening. Figure 37b is the resulting output for absolutely no inhibition, and does not appear to represent any sharpening. A better system for comparing

sharpness is needed, however. The pattern can be represented by a series of eight numbers with the highest number normalized to 100. The other seven numbers would then be percentages of the brightest line. The input distribution is then represented as 31,47,78,100,78,47,31,31 (zero sharpening) while the output for I=1.25E would be 0,0,0,100,0,0,0,0 (perfect sharpening). The normalized intensity distributions are indicated below each plot. Now it can be seen that there is some sharpening even with no inhibition at all. This is termed sharpening by threshold, and will be discussed later.

The point of this is to note that fairly useful sharpening results all the way down to I=0.156E. This surprizingly small amount of inhibition has a marked effect because it is actually enhanced by duplication. That is, as neurons U, V, and W generate strong outputs, each of them contributes to the inhibition of neuron X, thus reducing its normalized output count to 13 vice 44 for the no-inhibition case.

Another question that needs to be raised is that of how much sharpening is necessary to an organism. Certainly it cannot benefit from perceiving a world in which all contours have been converted into sharp lines. The answer to this question is unfortunately not available from these models. But perceptual studies generally do not seem to support sharpening as extreme as just demonstrated. Does this tend to show that perhaps inhibition, in real-life lateral inhibition networks, is low in magnitude relative to excitation? The answer is no, since the artificial character of the inputs just presented is responsible for the high degree of effectiveness of the network. In Fig 37a, the fact that all channels have their initial input immediately (fixed phase) is no coincidence. This was done to demonstrate the input style which produces the most striking

example of line sharpening. Other input styles resulted in a lesser degree of sharpening in some cases and in bizarre effects in other cases. Specifically, two other input styles were investigated: random, and constant frequency with random phase.

The results of line sharpening runs with random inputs are shown in Figs 40, 41, and 42. The number of inputs to each neuron was carefully chosen to be identical to the number for the constant frequency, fixed phase case. (This cannot be verified by manually counting markers in Fig 40a, since markers sometimes fall so close together so as to be unresolvable graphically. The counts were performed by computer and were verified to be identical to those of Fig 37a.) The striking difference between random inputs and constant frequency, fixed phase inputs is that much higher levels of inhibition are necessary if the random line sharpening is to even approach the latter. The reason is obvious: the constant frequency, fixed phase situation had a 100 percent chance that the correct channel would fire and thus inhibit first, and therefore maintain dominance. With random inputs, bunching can occur and force an output even in a strongly inhibited channel with low average input intensity.

Examining the normalized output distributions, it is clear that useful sharpening does not occur below I=1.25E for this style of random inputs, and the performance of the lateral inhibition network can be called marginal. The question arises as to what time sequence best represents what the actual inputs to a real-life lateral inhibition network might be. Considering that the stimulus is a bright line switched on at shortly before time zero, the fixed phase input is not totally unreasonable, since all receptors would begin to be stimulated at about the same time, and would see a steady level of stimulus and hence fire at an

unchanging rate. But there is certainly some degree of randomness associated with the process of quanta absorption, receptor pctential generation, and spike production. Additionally, saccadic eye movements would cause variations in the illumination a particualr receptor would get.

b. Extreme Phase Sensitivity

Continuing to pursue the best input representation, consider now a subject staring at a blank wall (Figs 43 and 44). If luminance were switched on at just before time zero, then randomness in absorption of quanta and development of the initial input is represented by the random location of the initial markers (Fig 44a). The fact that the subject is staring at a large wall of constant luminosity accounts for the fact that each channel gets the same input intensity. Saccadic eye movements would produce no effects in this example. Consider that once the first input is created, subsequent inputs are regularly spaced. The resulting outputs are bizarre, and demonstrate an extreme phase sensitivity. Basically, the first channel to generate an output in such a situation will maintain dominance in its locality. It is unlikely that this extreme phase sensitivity exists in nature.

c. Line Shift

If applied to the diffuse line display with random phase, this phenomenon can result in a channel with lower input intensity gaining and keeping dominance over a channel with higher input intensity (Fig 45). Note that channel Y maintains dominance even though it is lower in input intensity than channels U, V, W, and X. The competition between channels U and Y is of interest. Channel

U has the first output, but its inhibition of channel Y occurs during that neuron's refractory period, and is therefore ineffective. The inhibition on neuron U by the first output from neuron Y falls after the refractory period and is therefore effective. This effect indicates that the delay of spread of inhibition is important in spatial as well as temporal phenomena.

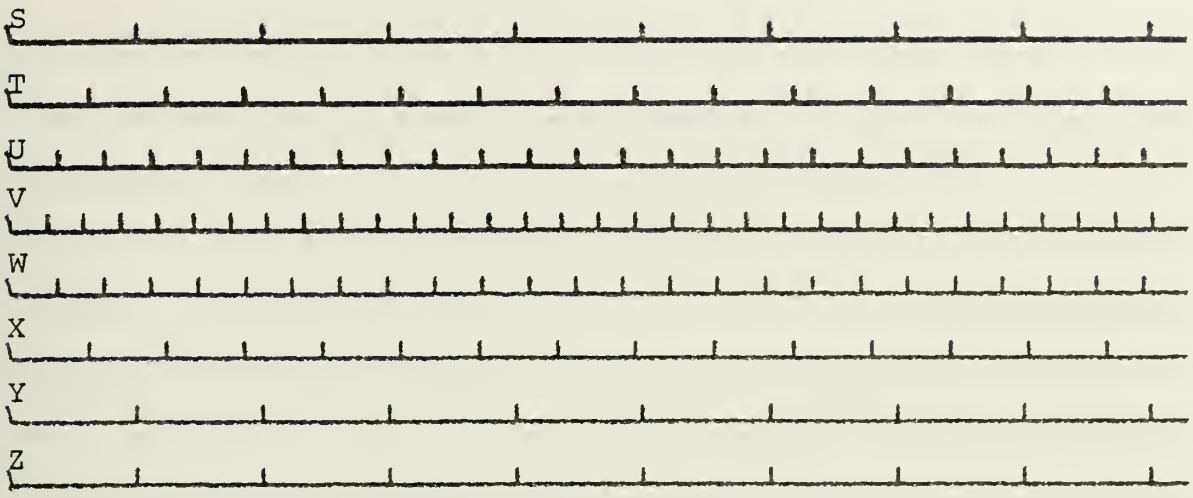
Line shift is undesirable for an animal trying to catch his dinner or avoid predators, and has undoubtedly been eliminated or minimized by randomization of inputs, optimization of spatial extent of inhibitory spread, or both.

d. Optimum Stimulus Representation

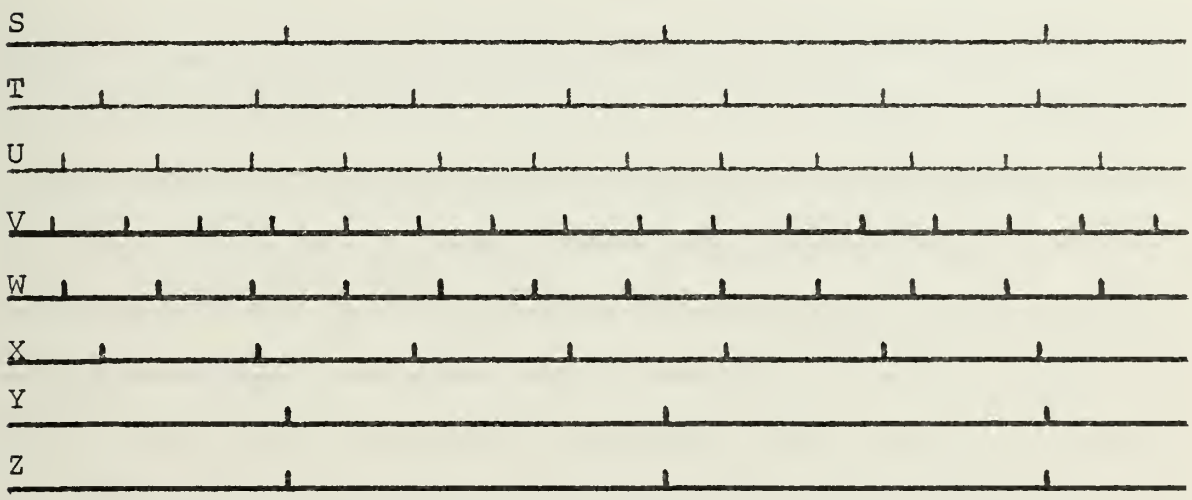
The payoff to this lengthy discussion of how to represent the inputs is the hypothesis that a new genre of random inputs needs to be devised. The random inputs of Fig 40a, generated by an algorithm described in Ref 5, have a Poisson distribution and seem to have too much bunching for the case of a subject fixing on a static visual field. The Poisson input distribution could be modified by a simple additional program which would make the input spacing somewhat more regular. Such a program would step through the poisson inputs and remove any marker found within a certain small time following any other marker. Additionally, the program would insert a marker if a longer increment of time were to elapse without a marker. The short and long time increments would be callable parameters.

e. Line Sharpening by Threshold

There is another conceivable mechanism by which



A. INPUT (31,47,78,100,78,47,31,31)



B. OUTPUT, NO INHIBITION (19,44,75,100,75,44,19,19)

C. OUTPUT, I=1.25E (0,0,0,100,0,0,0,0)

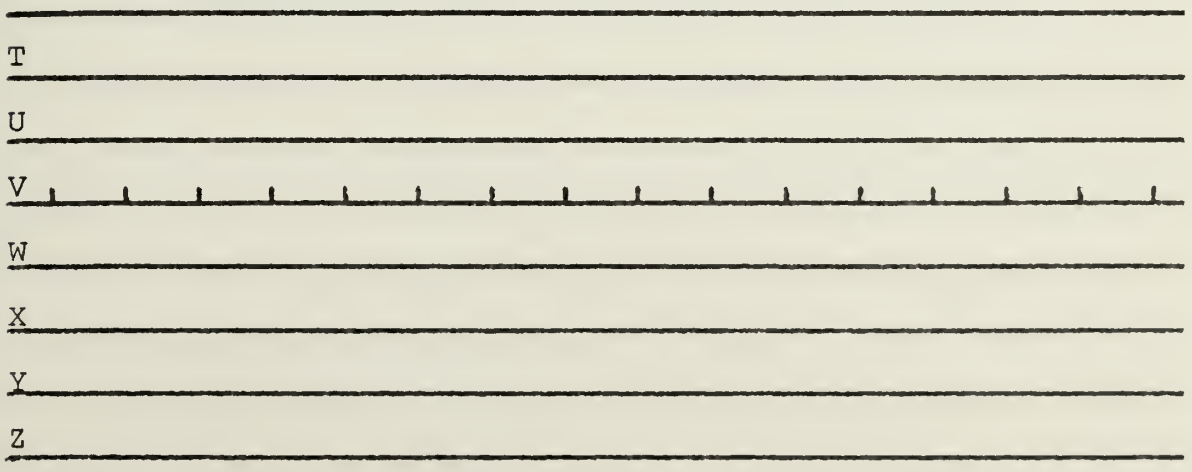
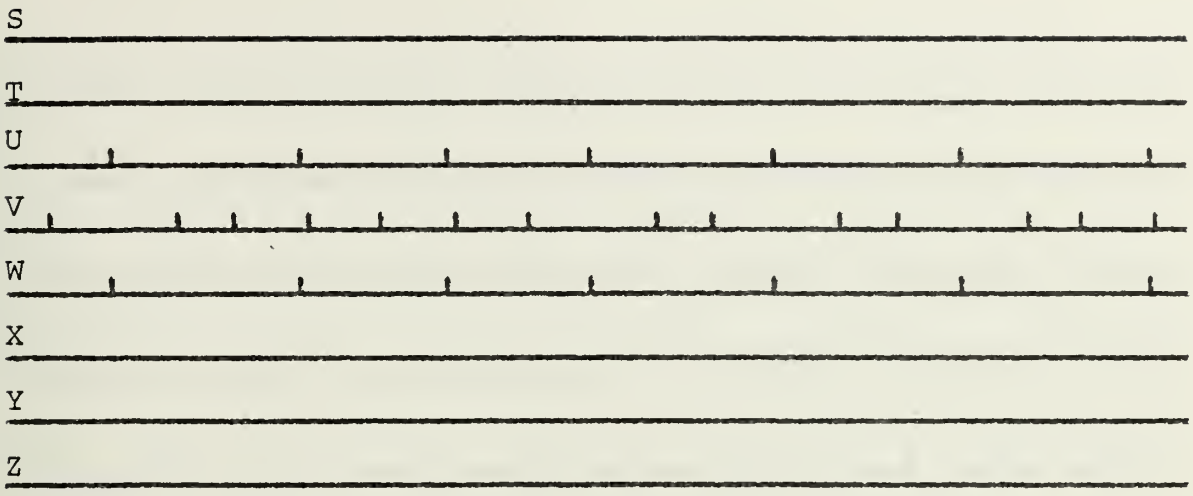
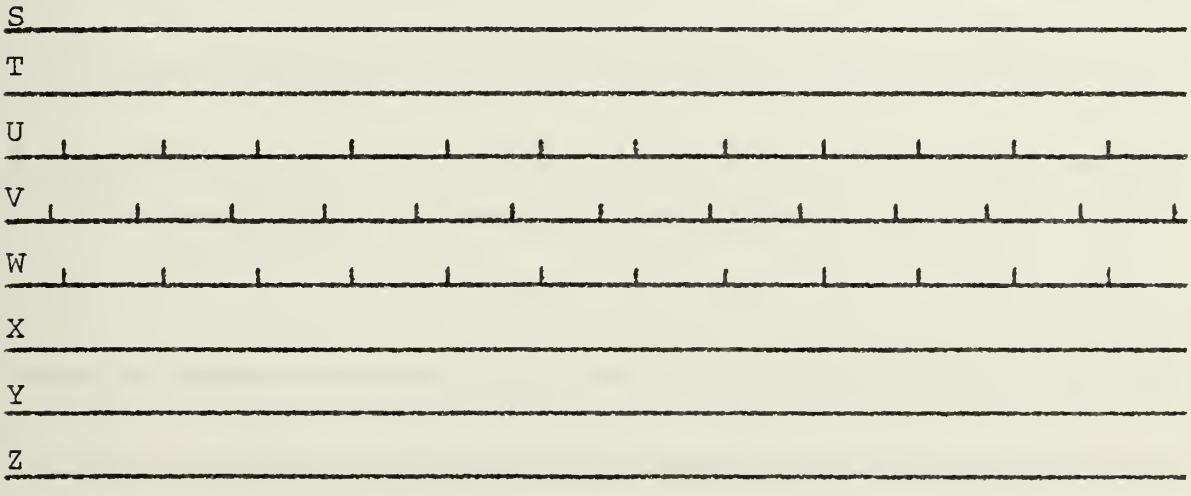


FIGURE 37 - LINE SHARPENING, REGULAR INPUTS, PART 1



A. OUTPUTS, I=0.625E (0,0,50,100,50,0,0,0)



B. OUTPUTS, I=0.3125E (0,0,92,100,92,0,0,0)

C. OUTPUTS, I=0.156E (0,13,80,100,80,13,0,0)

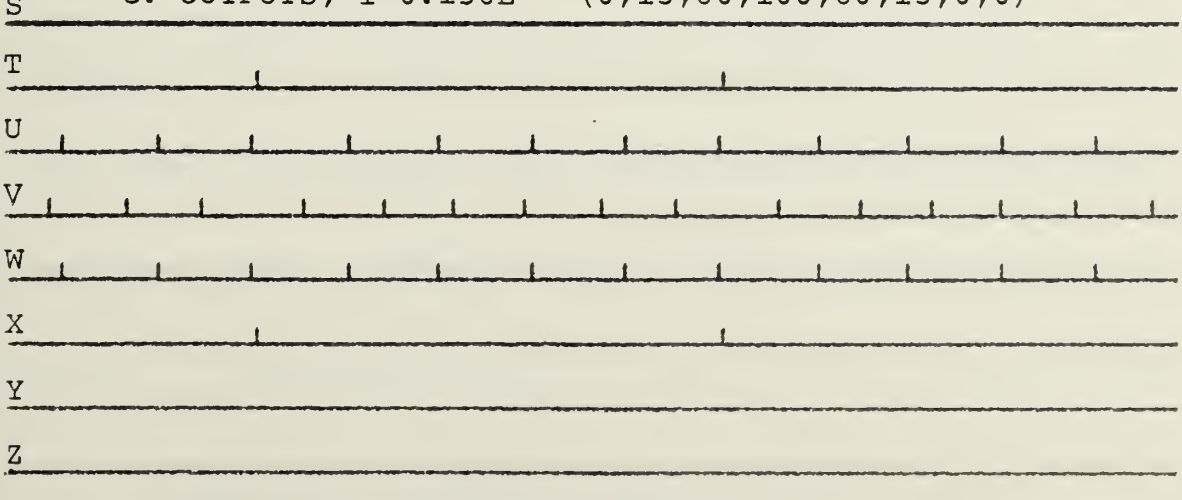
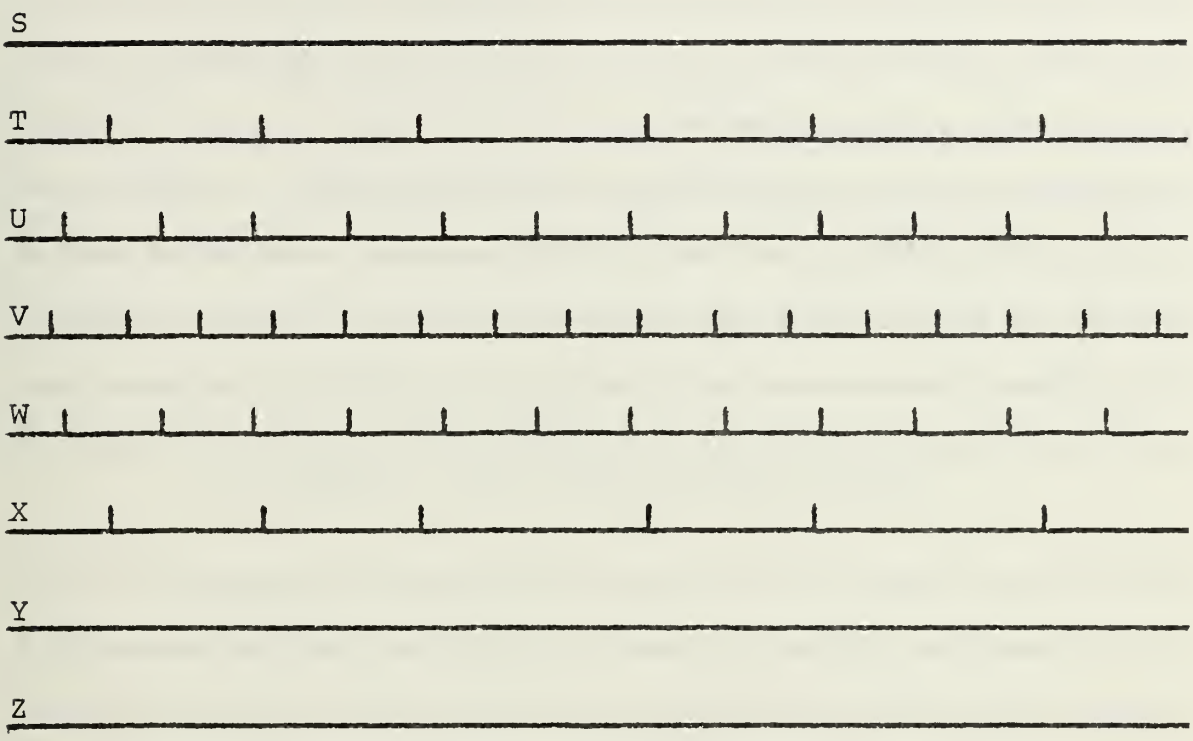
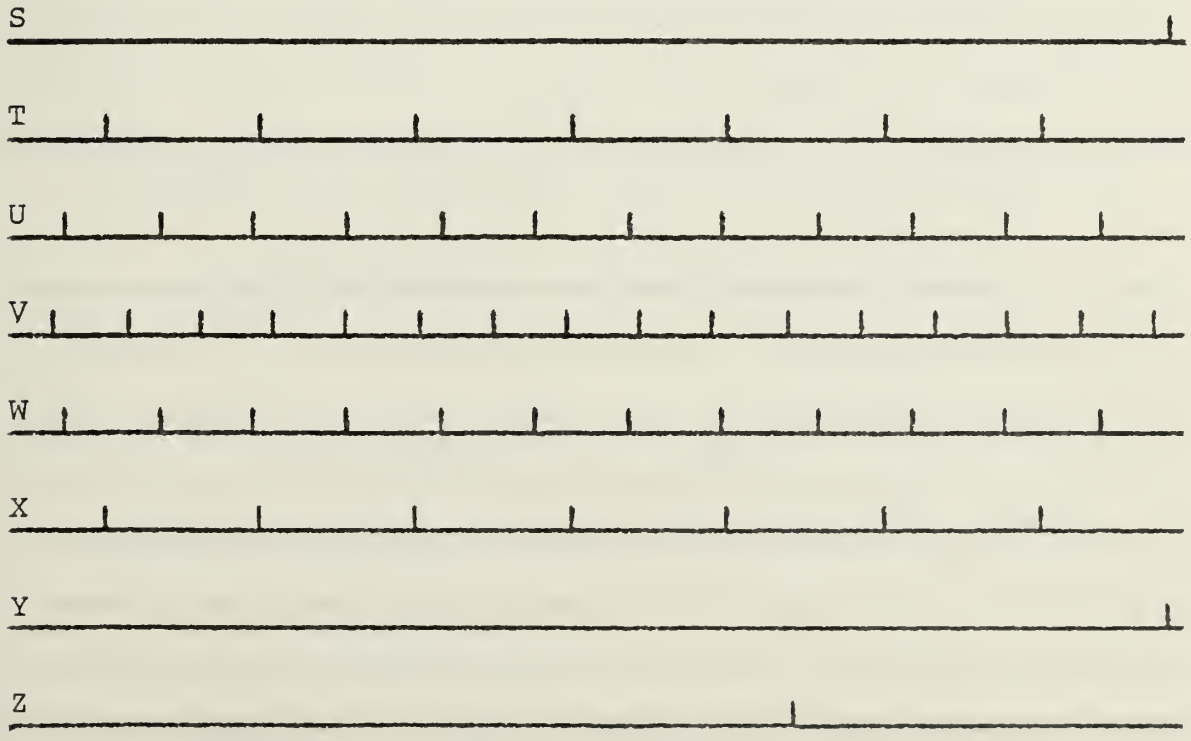


FIGURE 38 - LINE SHARPENING, REGULAR INPUTS, PART 2

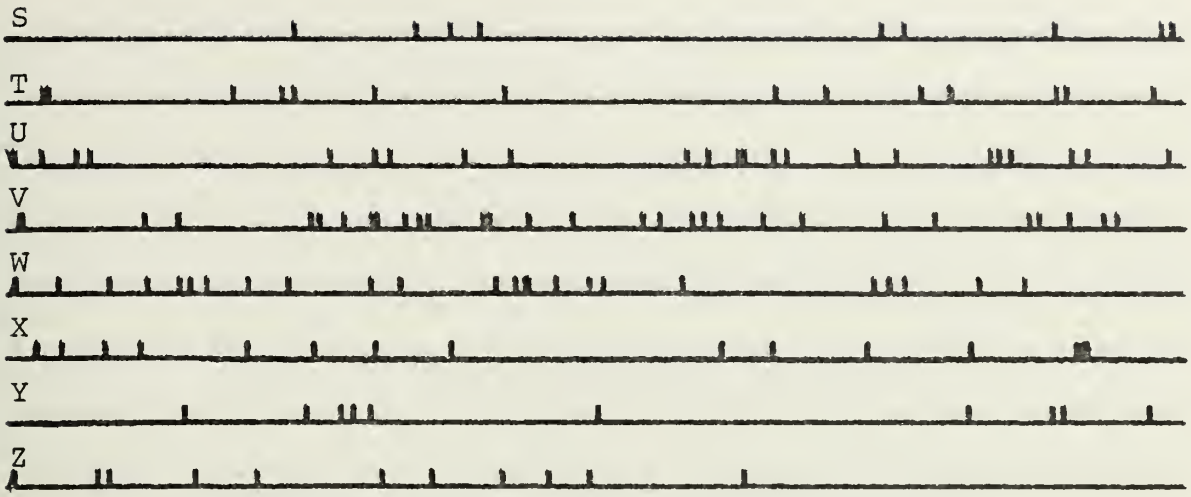


A. OUTPUTS, I=0.078E (0,38,75,100,75,38,0,0)

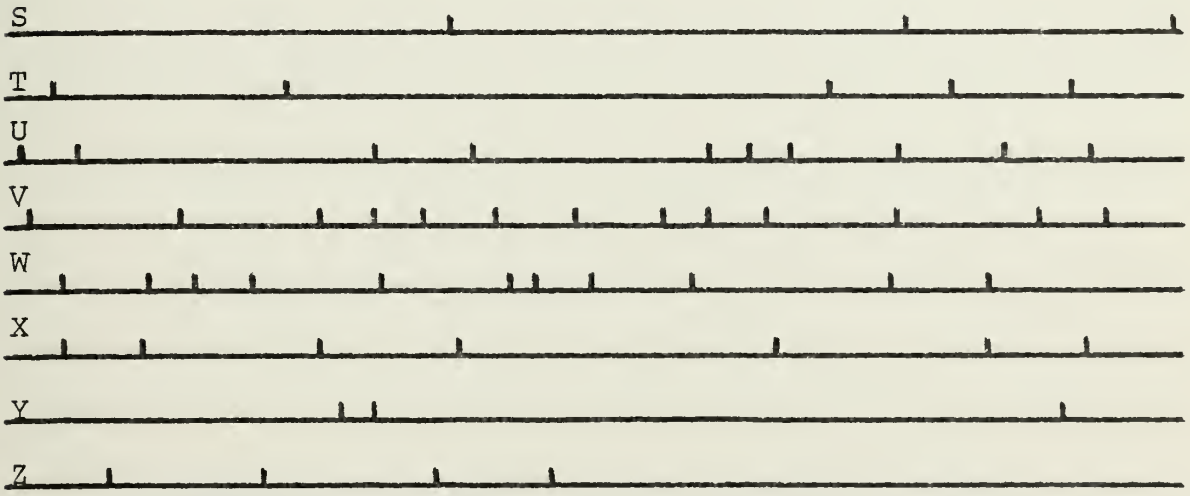


B. OUTPUTS, I=0.039E (6,44,75,100,75,44,6,6)

FIGURE 39 - LINE SHARPENING, REGULAR INPUTS, PART 3



A. INPUT (31,47,78,100,78,47,31,31)



B. OUTPUT, NO INHIBITION (23,38,77,100,85,54,23,31)

C. OUTPUT, I=5.0E (0,0,18,100,9,0,0,9)

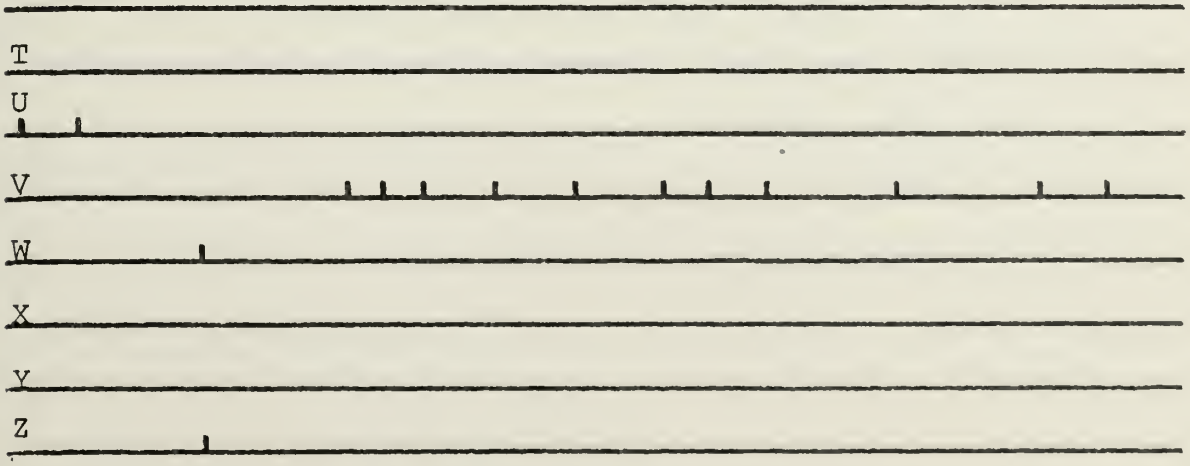
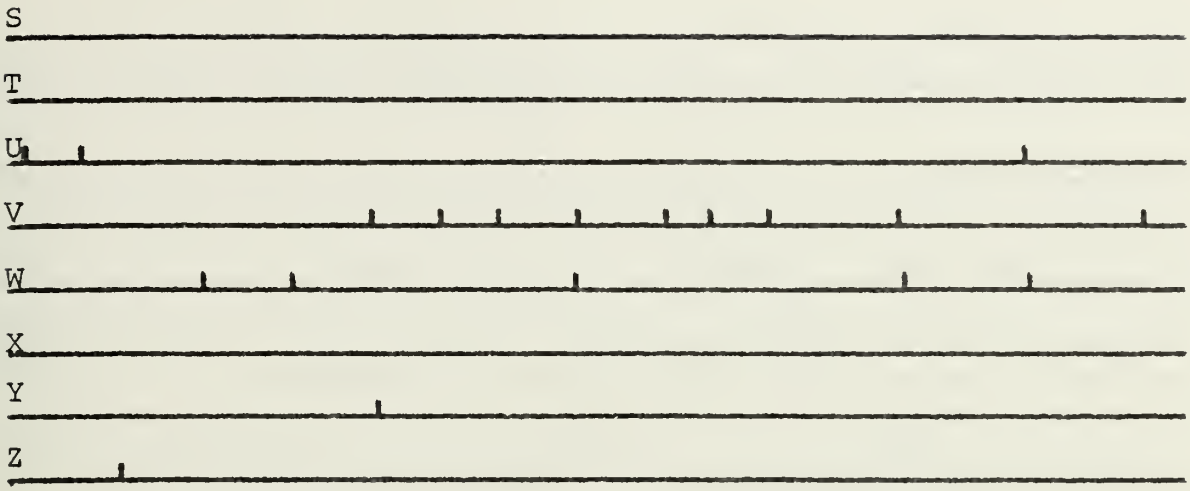
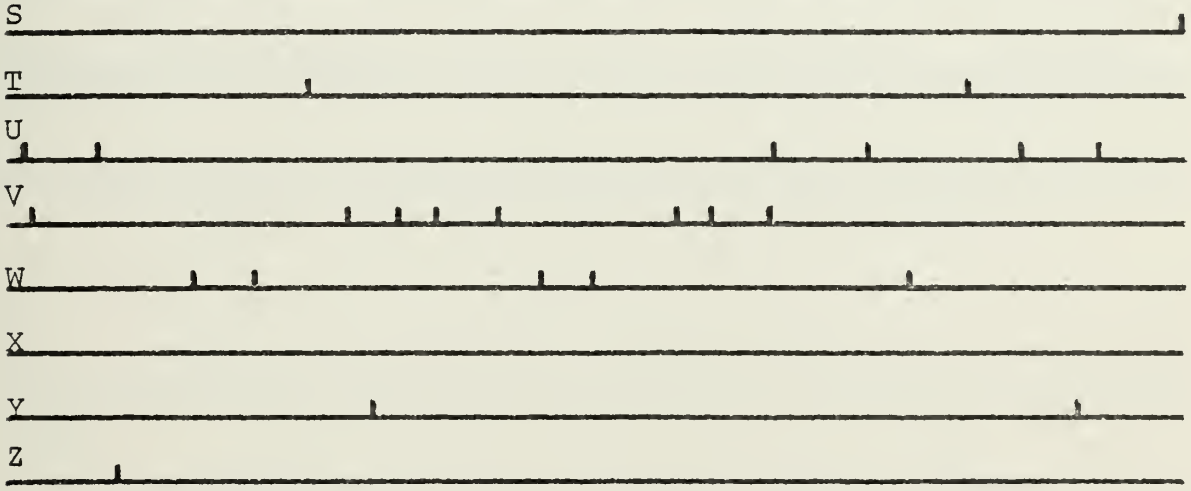


FIGURE 40 - LINE SHARPENING, RANDOM, PART 1



A. OUTPUTS, I=2.5E (0,0,33,100,56,0,11,11)



B. OUTPUTS, I=1.25E (13,25,75,100,63,0,25,13)

C. OUTPUTS, I=0.625E (20,20,70,100,50,20,20,20)

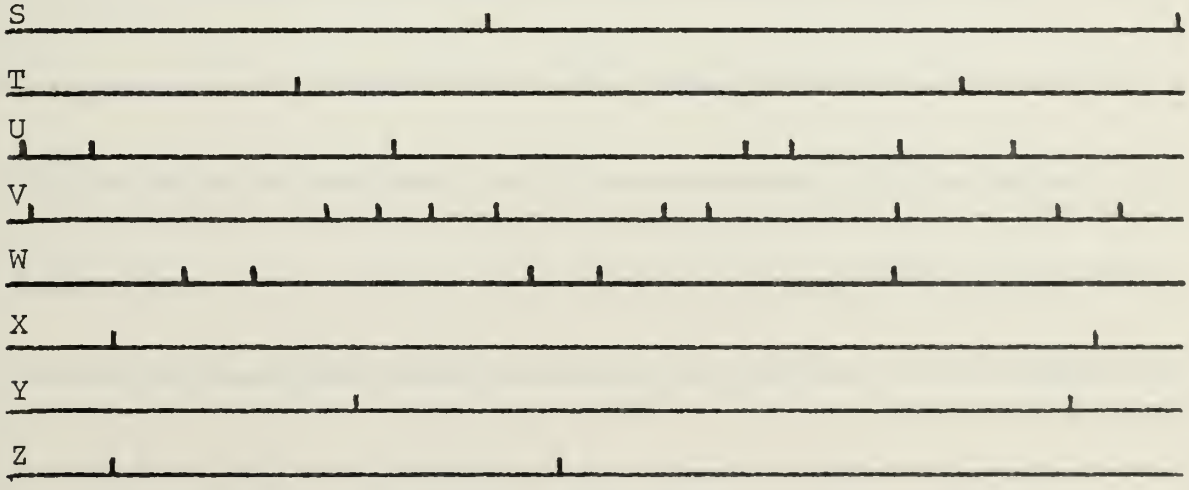
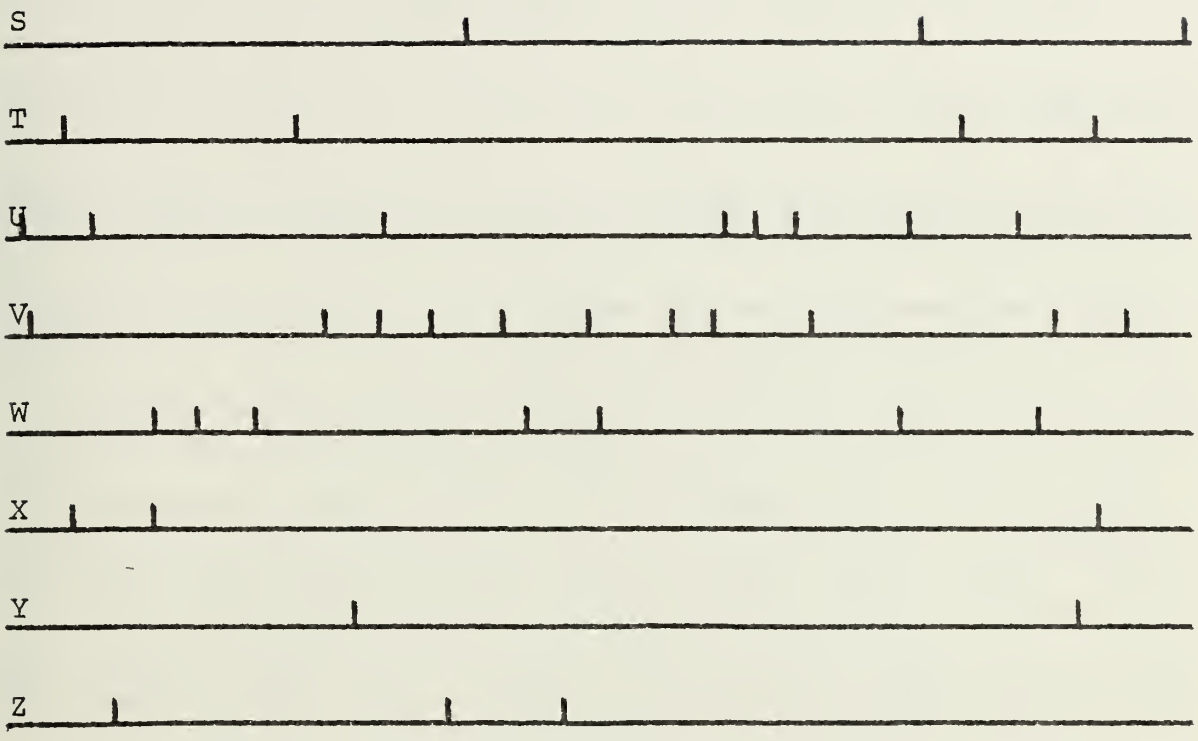
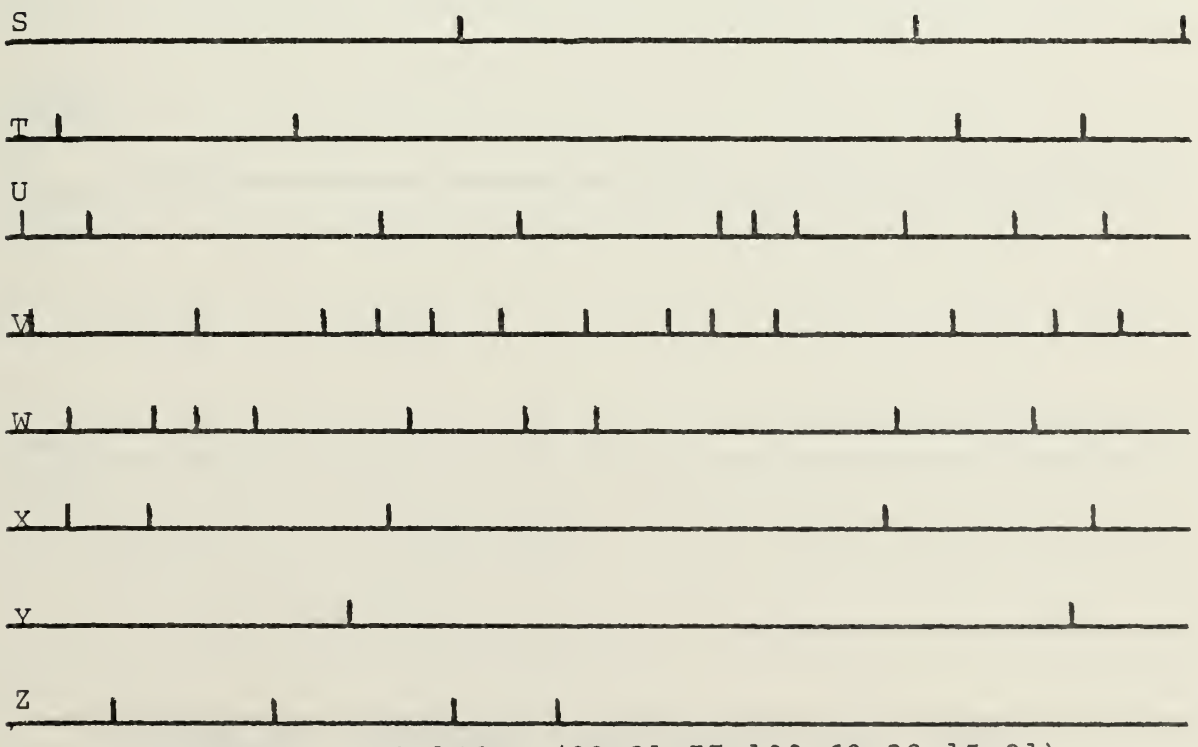


FIGURE 41 - LINE SHARPENING, RANDOM, PART 2

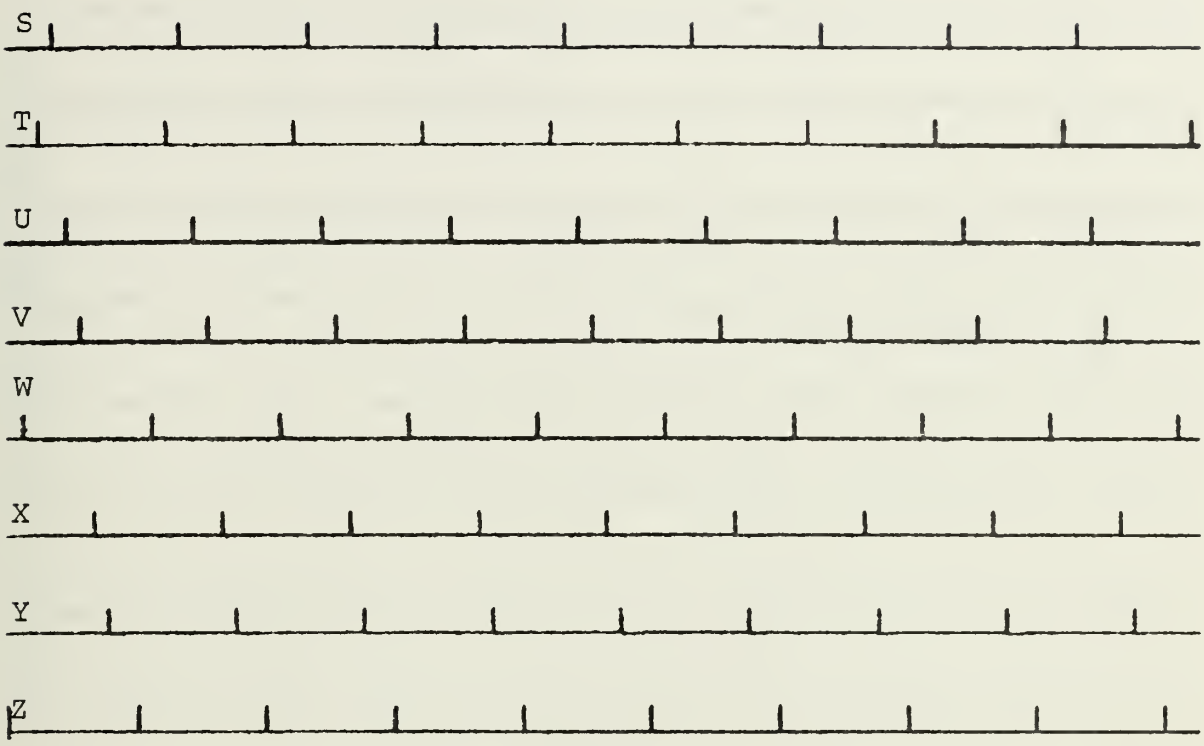


A. OUTPUTS, I=0.3125E (27,36,73,100,64,27,18,27)

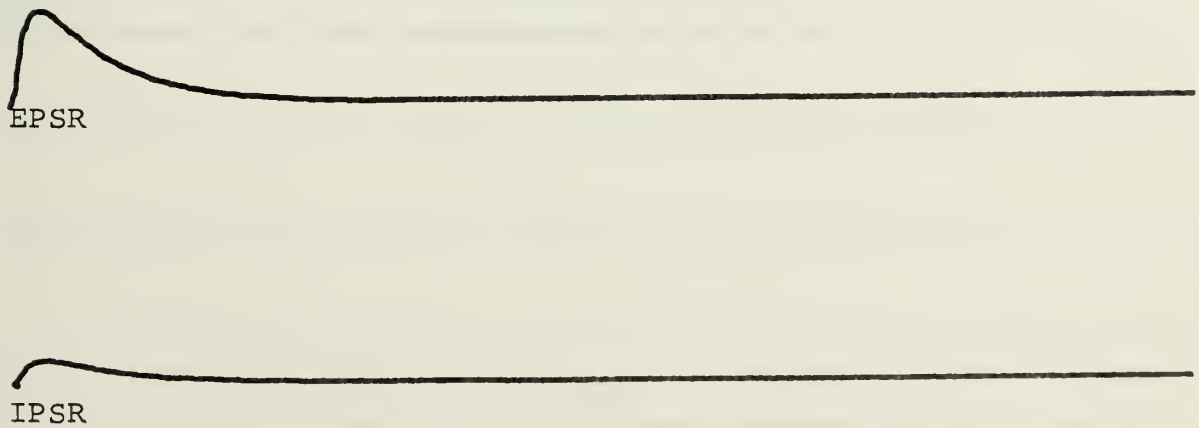


B. OUTPUTS, I=0.156E (23,31,77,100,69,38,15,31)

FIGURE 42 - LINE SHARPENING, RANDOM, PART 3

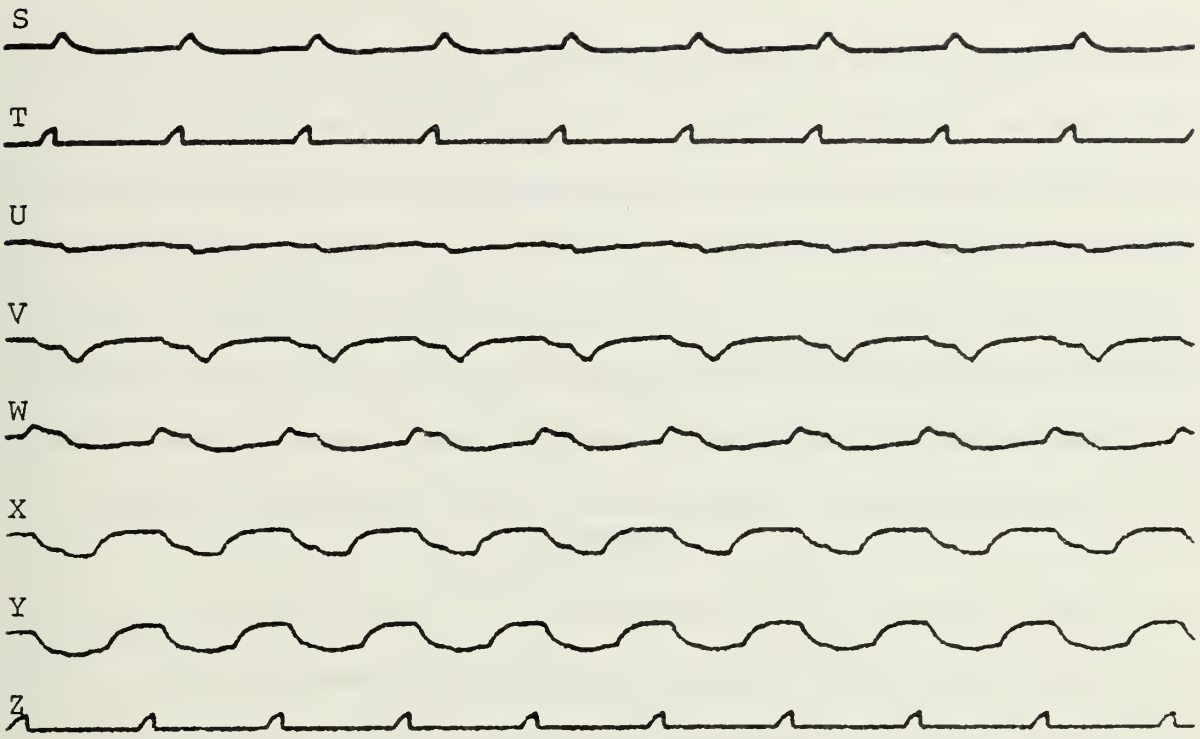


A. INPUT

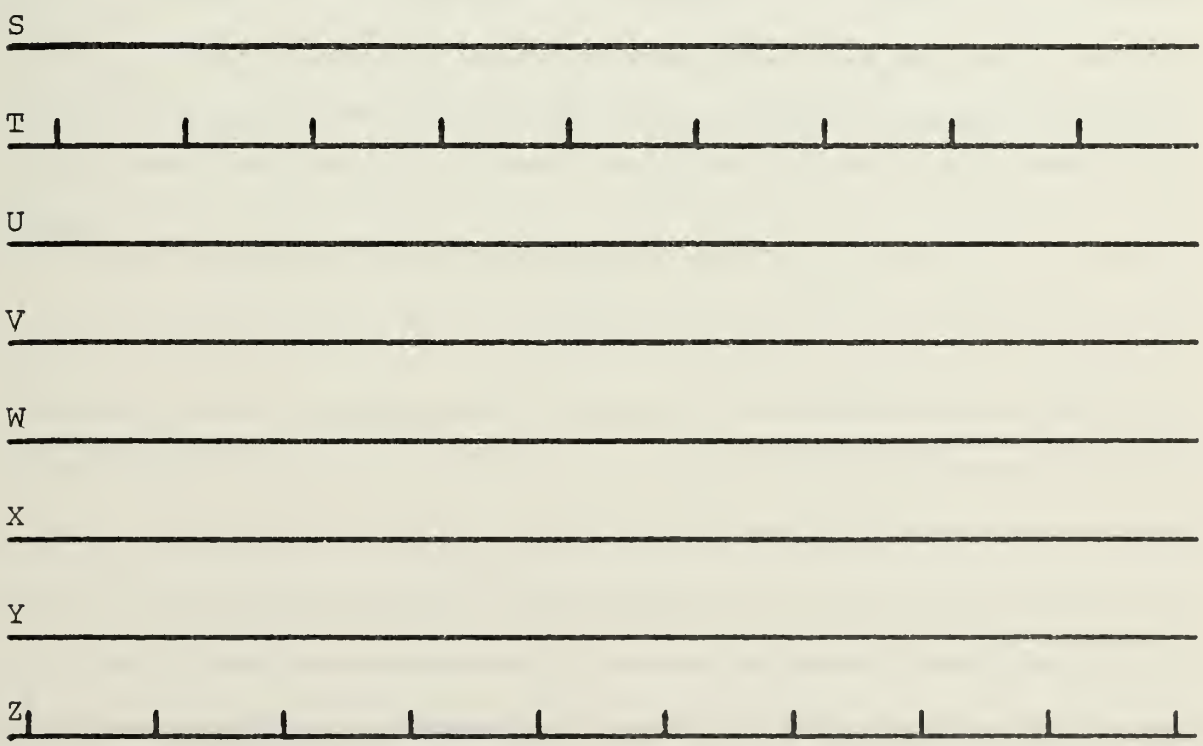


B. PSR

FIGURE 43 - ABNORMAL PHASE SENSITIVITY, PART 1

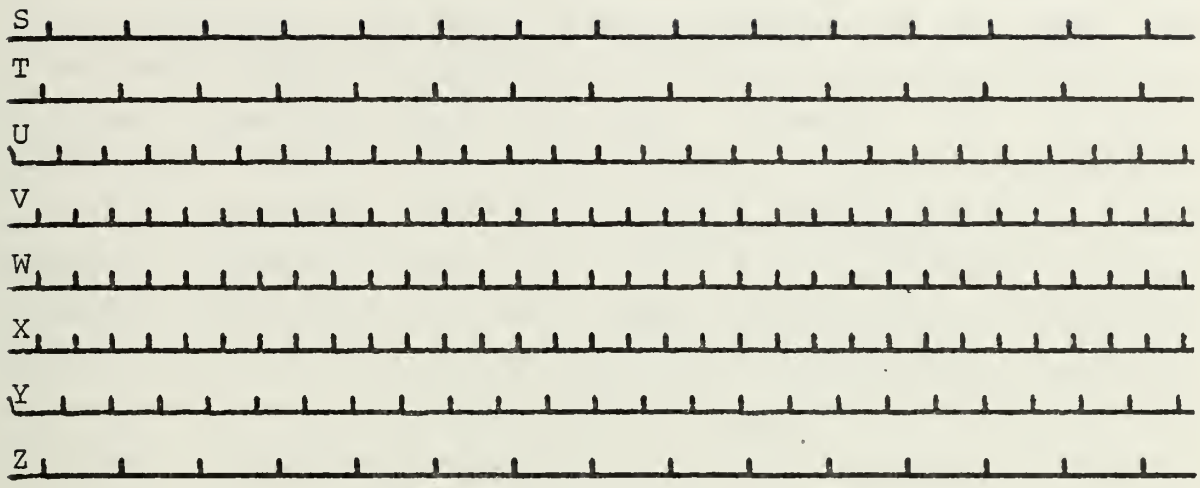


A. PSP

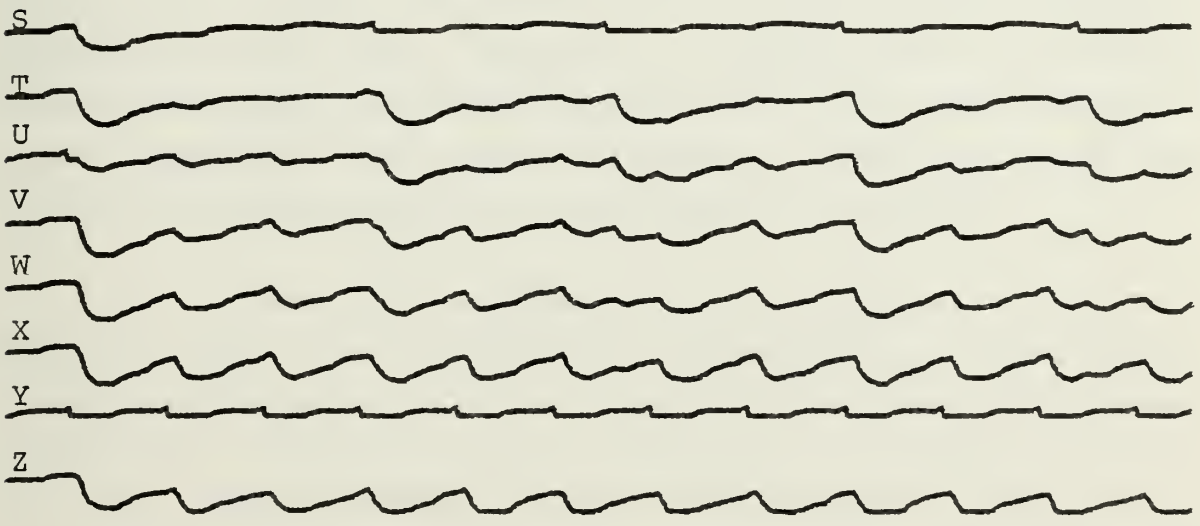


B. OUTPUT

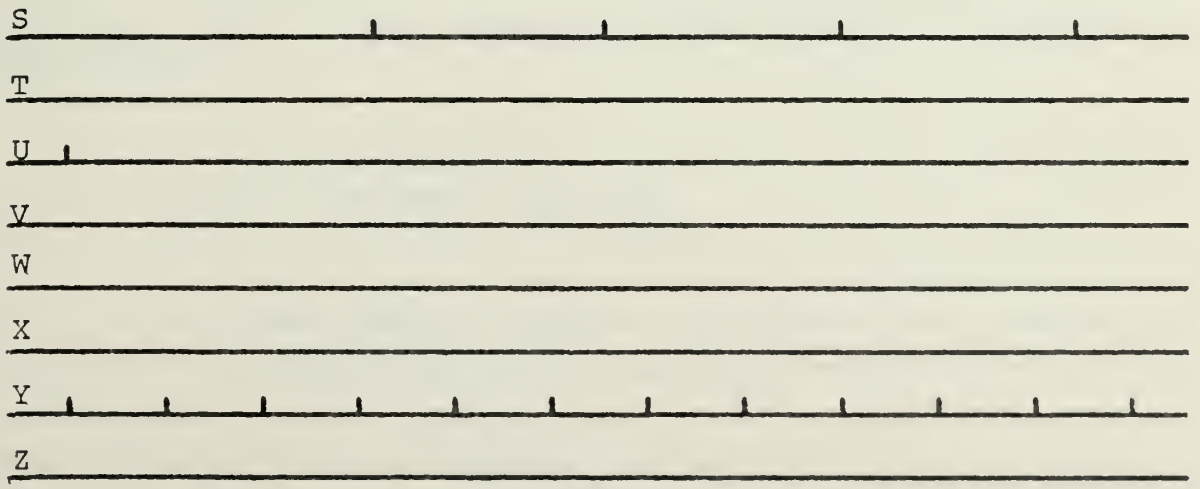
FIGURE 44 - ABNORMAL PHASE SENSITIVITY, PART 2



A. INPUT



B. PSP



C. OUTPUT

FIGURE 45 - LINE SHIFT

line sharpening could occur. By adjusting the threshold for spike production, the most intense channel can be made to fire more often in proportion to its inputs than the less intense channels, even in total absence of lateral inhibition. Such a mechanism may very well be in operation in real organisms, but it is less effective than the powerful lateral inhibition, and also considerably less flexible. That is, there are many parameters in lateral inhibition which are highly variable in the modeling program as well as in the organism, and all involve, in one way or another, different schemes of neural interconnections. Because of the immense number of neurons available, and the staggering number of connections a single neuron can make, parameters which vary because of differences in neural interconnections are realistically capable of wide latitude. It is improbable that spike threshold variations would be capable of producing such a wide range of effects.

Threshold sharpening can be discerned in Fig 37. Notice that the normalized distribution goes from 31,47,78,100,78,47,31,31 to 19,44,75,100,75,44,19,19 when inhibition is removed. This is strictly a threshold effect. In Fig 40, however, the distribution goes to 23,38,77,100,85,54,23,31, which can hardly be considered sharpened. It appears that the bunching of inputs in the random input has decreased the effect of threshold sharpening.

4. Spatial Frequency Response

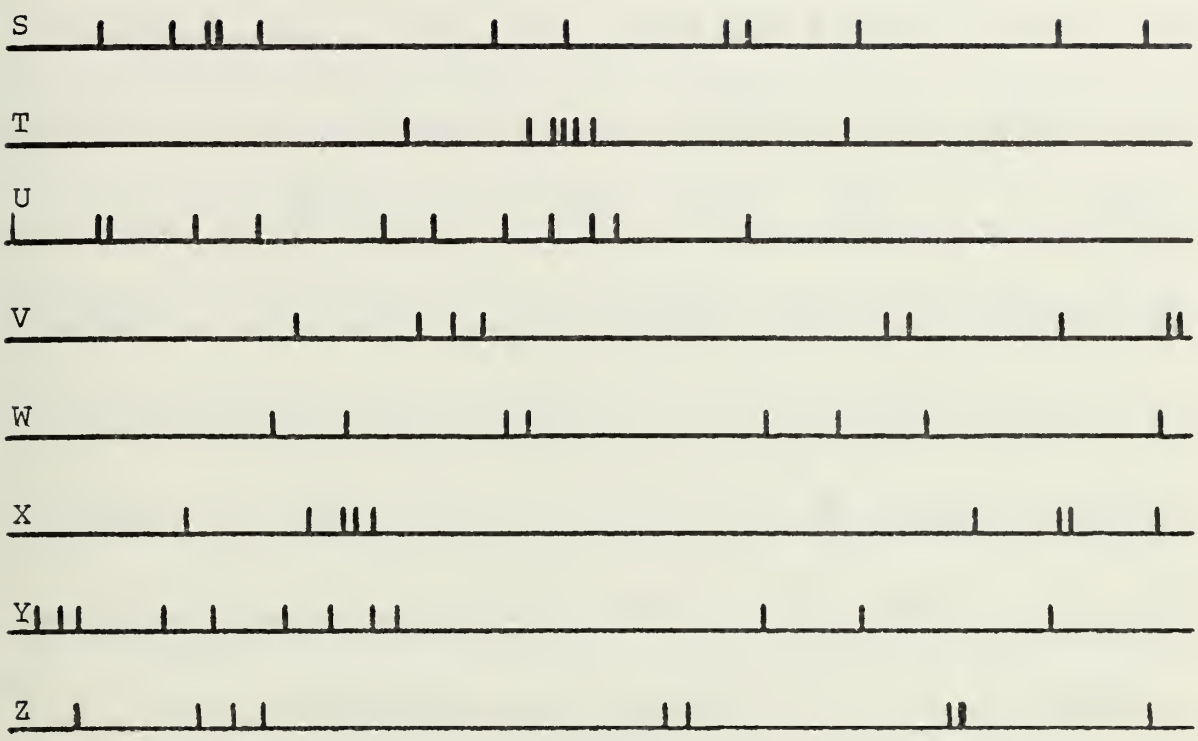
With only eight channels available for inputting spatial distributions, any meaningful verification of the modulation transfer function (page 26) is nearly impossible. Two effects relating closely to spatial frequency response will, however, be demonstrated. These are, first, the

suppression of steady, uniform levels of illumination (representing a low spatial frequency) and second, the enhancement of contrast at a border between two regions of uniform intensity (Mach bands).

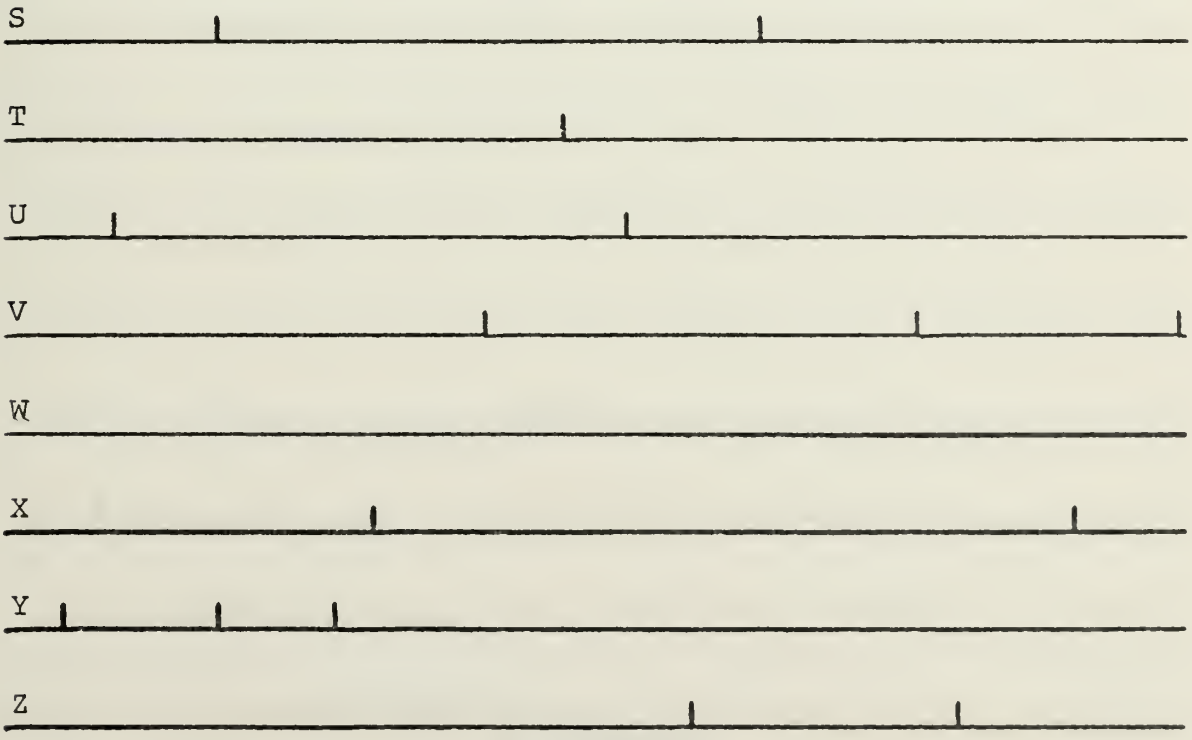
a. Suppression of Low Spatial Frequencies

The enormous dynamic range of the visual system for light intensity is the result of several factors. First, there is the fairly minor effect of change in pupillary aperture size, which occurs in a time frame of several minutes following a step change in light intensity. Second, there is the major effect of rhodopsin concentration changes, which occur over a thirty minute time period. Third, there is the logarithmic response of receptor cells. Last, there is the effect that an inhibitory neural network should cause an enhancement of dynamic range by virtue of its low response to low spatial frequencies [Refs 6 and 17]. That is, as few inputs arrive, few outputs occur, and few inhibitions result. As more inputs arrive, outputs increase, but so do inhibitions, thus holding down the output level. As in regularization by a lateral inhibition network, the duration of the IPSR seems to be a key here, since for this amount of time, outputs are unlikely. As inputs increase in number, they are more likely to meet with a large hyperpolarization.

LINHIB allows quantitative exploration of this effect (Figs 46 and 47). With random inputs totaling 83 (dim light), the inhibitory neural network produces 15 total outputs. If the total number of inputs is increased to 235, a factor of 2.83 increase, a total of 41 outputs are produced, a factor of 2.73 increase. This result was quite disappointing, as the latter number should have been much smaller in order to account for any noticeable effect in

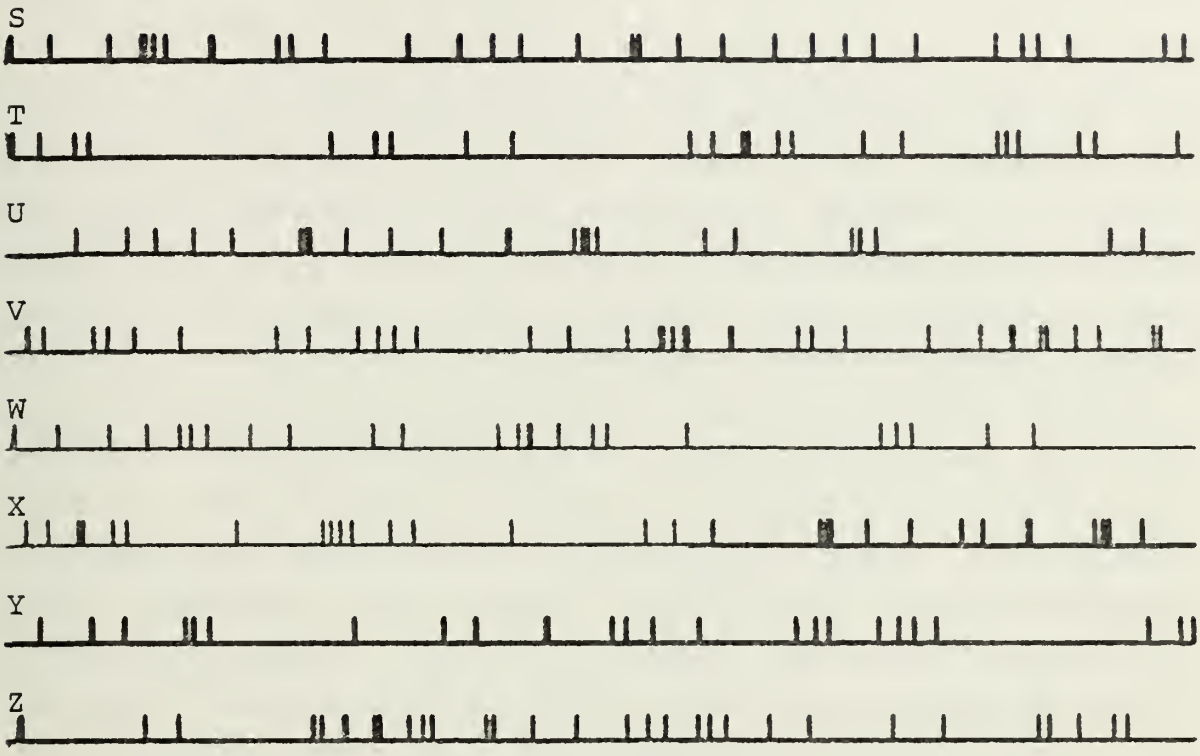


A. INPUT

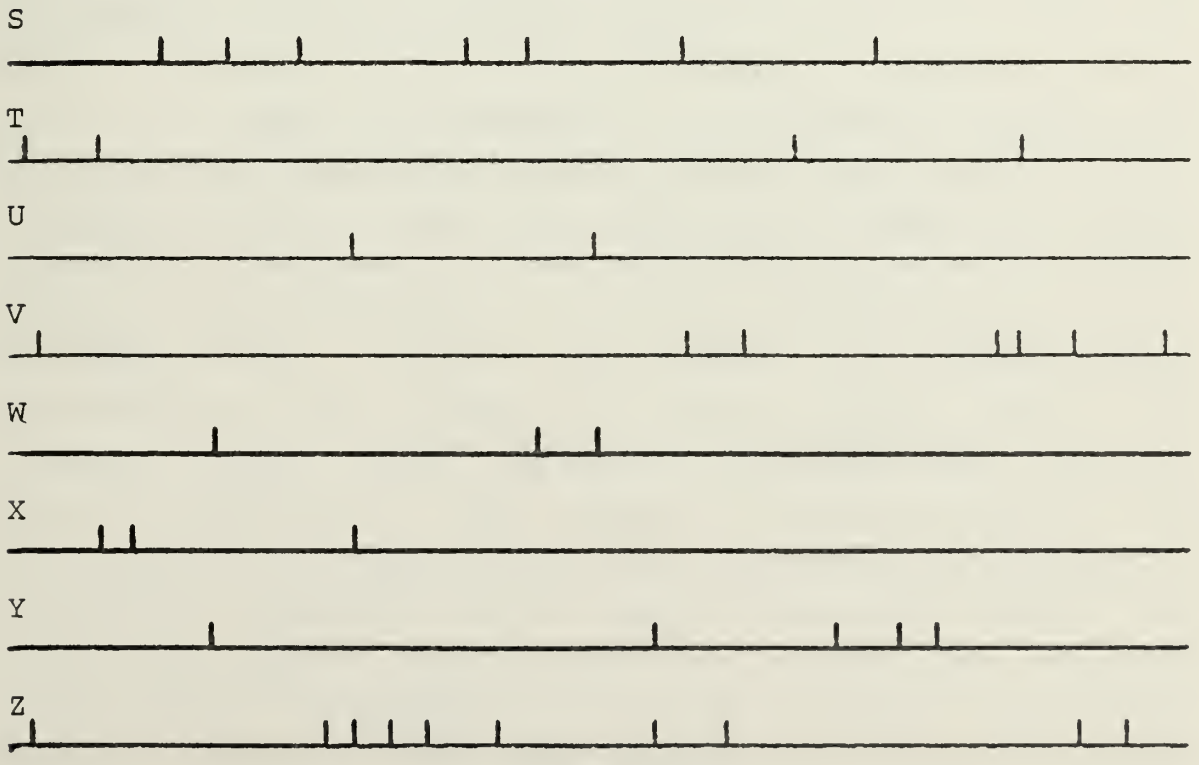


B. OUTPUT

FIGURE 46 - LOW SPATIAL FREQUENCY RESPONSE, DIM



A. INPUTS



B. OUTPUTS

FIGURE 47 - LOW SPATIAL FREQUENCY RESPONSE, BRIGHT

visual perception.

While this dynamic range enhancement may appear to be only slight (or indeed, questionable), many factors must be considered. First, this effect is very sensitive to parameter selection (as are most effects in neural modeling). The process of natural selection/evolution over millions of years would probably have produced a more effective combination of parameters in the visual system than can be happened upon in the laboratory during modeling. Second, with only eight neurons in the array, we are not dealing with a true zero spatial frequency, but rather with a bright band on an occult background. Third, the LINHIB model provides for lateral inhibition to occur whenever there is an cutput. There is evidence that in limulus, a threshold for inhibition exists. That is, if outputs are at a low temporal frequency, no inhibition would occur, but above a certain threshold, inhibition would begin to cccur. This would cause an increase in the dynamic range by increasing the output for dim inputs.

Thus the attenuation of low spatial frequencies by a lateral inhibition network has not been proven by this example. To the contrary, the 2.83 fold increase of input intensity producing a 2.73 fold increase in outputs casts doubt on the suppression of low spatial frequencies at all. In fact, even a single EIPSP neuron, when presented with successively more intense excitatory inputs, will not produce outputs which increase by the same ratio [Ref 5]. This is because as the number of random inputs are increased, any of them which occur very shortly following an output are "wasted", since inputs occurring during the refractory period have no effect on the PSP. Thus the very slight observed suppression could have resulted from this effect, and not from and characteristics of the lateral inhibition network. It became apparent that there are two

sides to the EIPSP neuron's output/input ratio story. If the number of inputs is initially very low (such as six per channel), the ratio will be low, since inputs tend to occur alone. As the number increases from this very low value, the output/input ratio increases as inputs occur closer together, temporal summation coming into play. A 4.13 fold input increase producing a 3.13 fold output increase is now seen to be much more impressive, since it represents a large amount of hidden suppression in overcoming the temporal summation effect in the neuron output/input characteristic.

More runs were made to attempt to prove or disprove the existence of low spatial frequency attenuation. A new pair of input fields was used, this time temporally random but spatially very uniform at six inputs per channel for the dim and 31 inputs per channel for the bright field (a factor of 4.13). After many hours of parameter variation, the best performance attainable was a 3.13 ratio of outputs, which is still not overly impressive. It was decided to determine what possible offsetting effect due to the EIPSP neuron input/cutput characteristic might be present, so the same runs were made without inhibition. The result was surprising in that a 4.13 fold increase in inputs produced a 5.94 fold increase in outputs without inhibition. This makes the low frequency attenuation of the lateral inhibition network more impressive.

One possible source of poor performance in attenuation of low spatial frequencies might be that a uniform field over an array of only eight neurons is too small to be considered a low spatial frequency. In order to attempt to eliminate this effect, LID=111111101111111 was used. As will be mentioned in the next section, this LID tends to give the effect of an infinite array. The result was disappointing: a 4.13 fold increase in input intensities produced a 3.6 fold increase in output intensities, which

was not as good as other runs with ramp type LID's.

This investigation should be continued following the develcpment of the improved random inputs discussed earlier. It is also probable that the use of several LINHIB processing stages in series could improve the suppression of lcw spatial frequencies.

Perhaps the most impressive quality of the retina's lateral inhibition network is that it allows viewing of a large bright region and its low contrast details in one area of the visual field simultaneous with a large dim region and its low contrast details. The retina, with its ability to adjust its overall sensitivity, is the living analogy to a photographic film which has an automatically adapting speed. Therefore, because of the ability cf the retina to have different sensitivities at different regions, a truer analogy would be a photographic film which would adjust its sensitivity locally to enhance contrast at every point in the field of view [Ref 17].

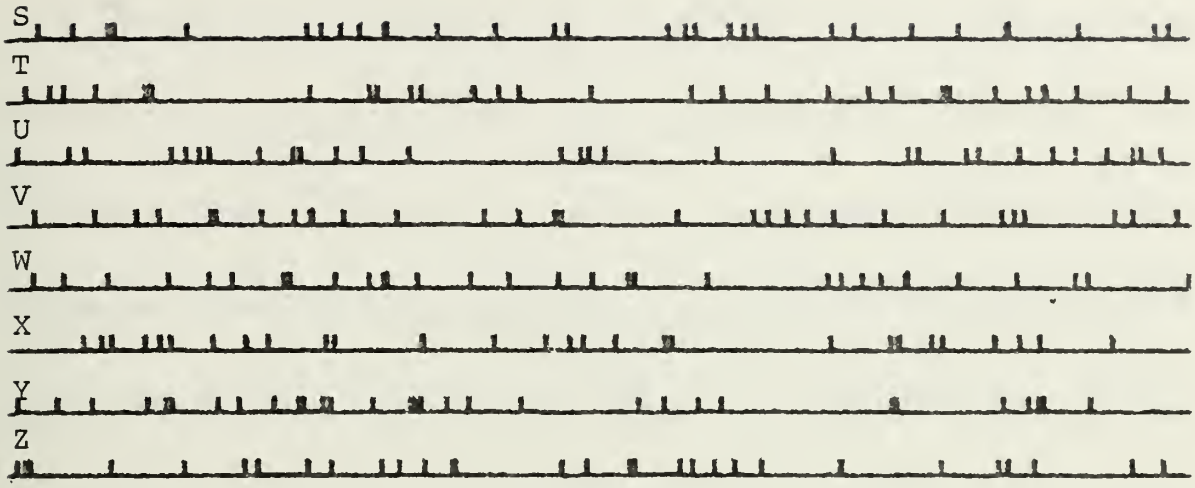
b. Mach Bands

When a bright region borders on a dim one with a relatively sharp contour separating the two, the visual system's spatial frequency response sharpens the contrast at the border. This appears to the observer as a darkening of the dark region near the border, and a lightening of the light region near the border, the so-called Mach bands. Consider the input field of Fig 48, which represents a bright bar on a very dark background. The outputs do not have the same distribution as tne inputs, and represent a crude Mach band phenomenon. Specifically, the input distribution is flat while the output is 83,42,42,58,33,75,8,100. Recalling that the eight neuron

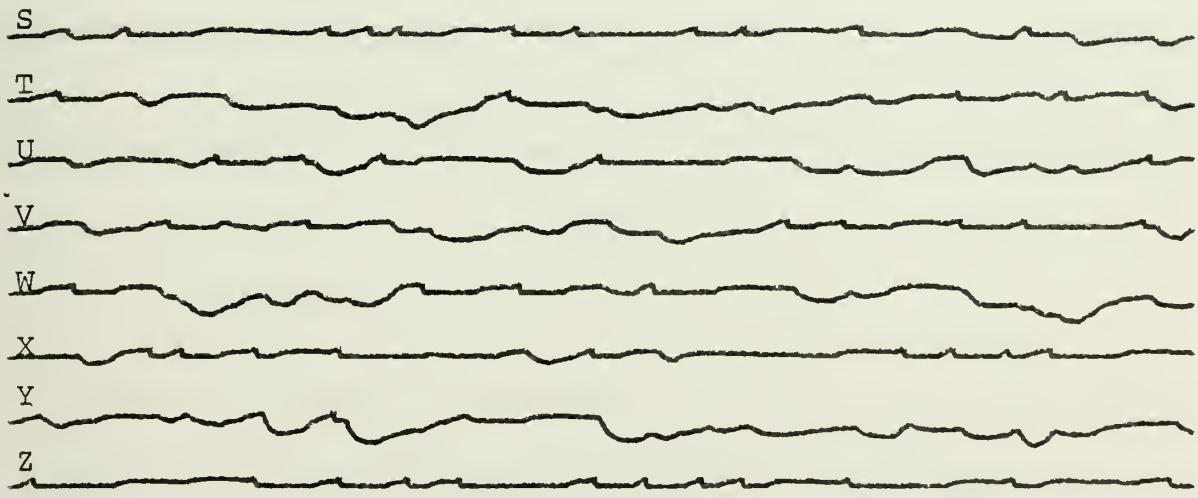
array really represents eight neurons receiving some degree of illumination while all bordering neurons are in total darkness, it is clear that the edges of the distribution (neurons S and Z) should appear lighter if the phenomenon is to be present. They do indeed appear lighter.

The LID was chosen as 000000101000000 for this demonstration in order to enhance the effectiveness. In order to understand this, consider why Mach bands appear. Central neurons (T, U, V, W, X, and Y) receive double inhibition (once from each immediate neighbor) whereas edge neurons (S and Z) receive only single inhibition (none from above S or below Z). By choosing LID=111111101111111 the phenomenon can be made to vanish, since each neuron is inhibited by each of the other seven (Fig 49). Specifically, the output distribution is 83,100,100,100,83,100,83,100, which is nearly flat, and certainly show no prominent Mach bands. For this small network, this LID represents infinite spread of inhibition, and tends to model an infinite array of neurons (so long as the distribution is uniform).

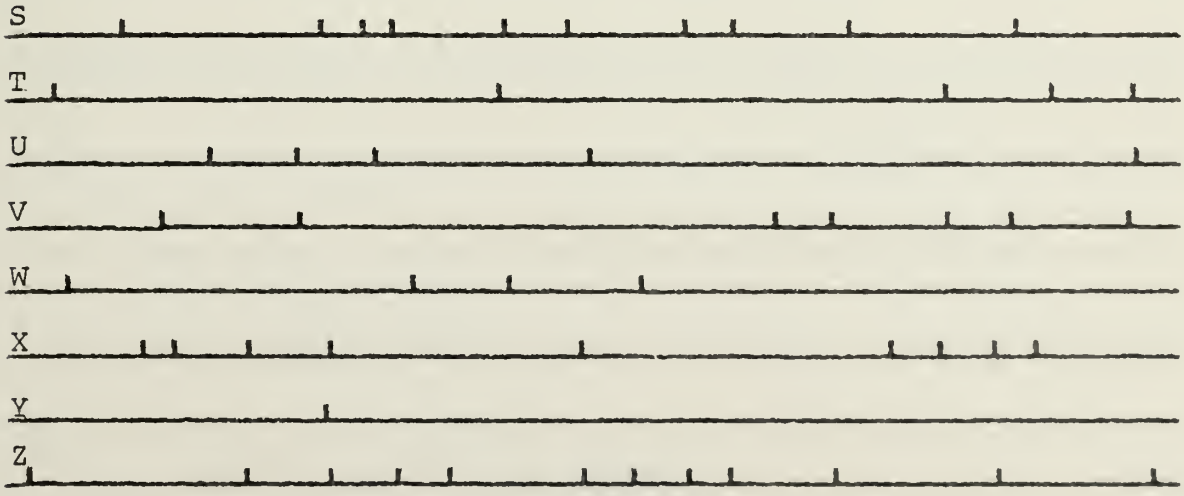
Some thought suggests that the most effective contour sharpening will result if the dimension of spread of inhibition is on the same order as the dimension of the contour.



A. INPUT

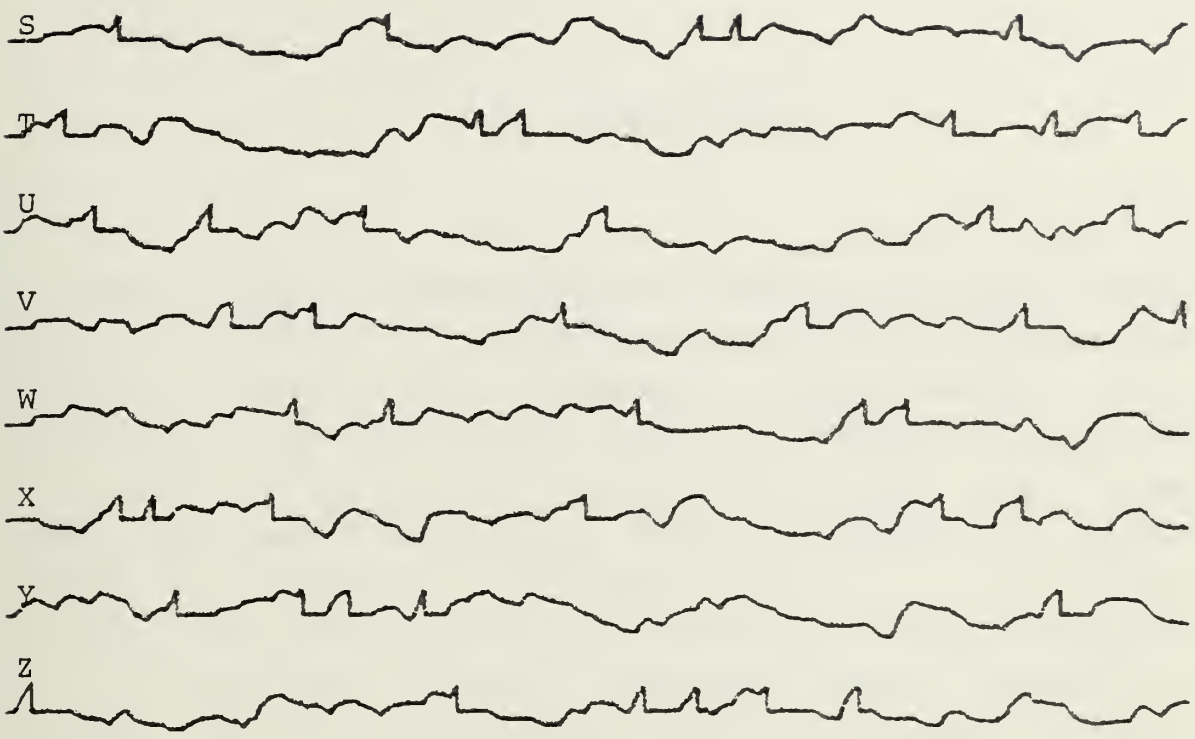


B. PSP

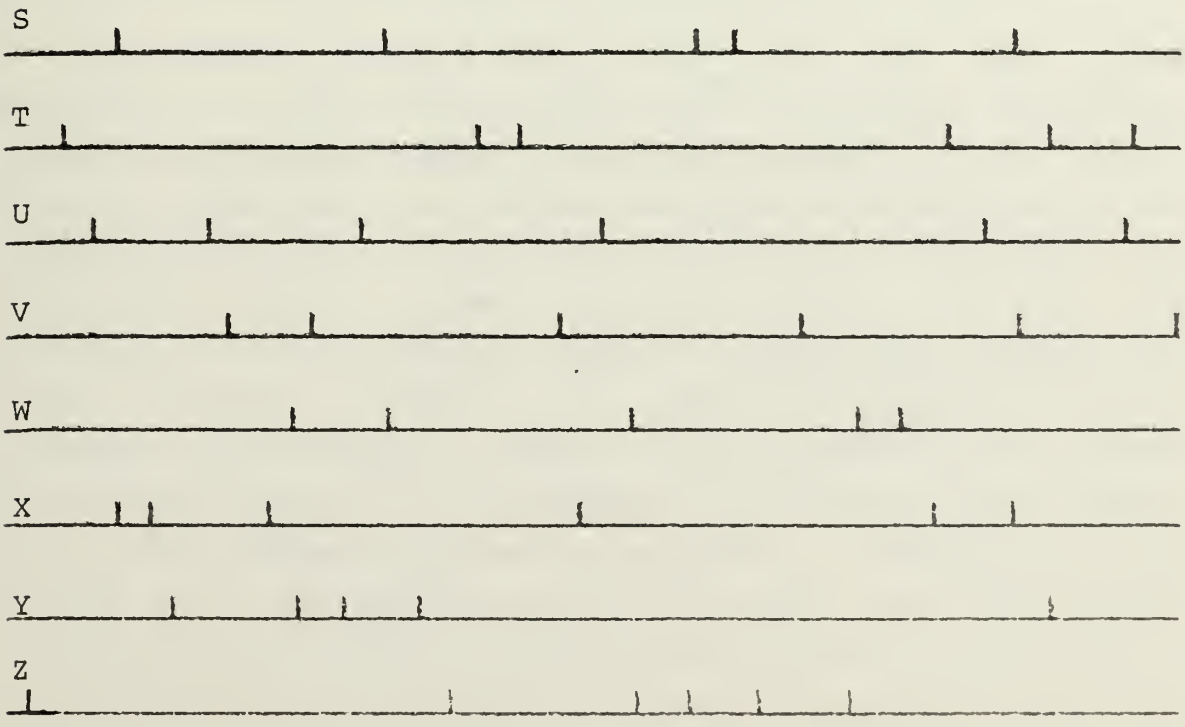


C. OUTPUT

FIGURE 48 - MACH BANDS



A. INPUT



B. OUTPUT

FIGURE 49 - ABSENCE OF MACH BANDS

V. CONCLUSIONS

The concept of target neurons which contain PSP's which vary from a period of hyperpolarization to a period of relative depolarization is hinted at by Refs 2 and 4. This concept has been used in this thesis to model a speed band pass gate as well as to regularize a random input spike train. The neural oscillator, used to drive the second neuron to generate a sinusoidal PSP, is a very simple circuit, and should therefore be sought in living organisms. The location of a neural oscillator would probably be central, since this is where the target neuron for the band pass speed detector is located. Both of these models work very well, and should be considered as good possibilities as to how such phenomena might actually occur in nature.

The models presented herein for slow and fast speed detectors utilize the concept of a lateral inhibitory network performing temporal functions rather than the usual spatial functions. By varying the IPSR duration, extent of inhibitory spread, and delay in spread of inhibition, an effective neural speed detection system emerges. The location of such a circuit should be addressed, since in the model, ganglion outputs are summed on a geniculate cell, whereas in the rabbit, a single ganglion cell is the target neuron [Ref 1]. But it is probably also true that the rabbit has a later stage of processing wherein a geniculate cell serves as a target for speed detection. With this slight reservation, the model is believable as well as effective.

The scheme used in this thesis for establishing null versus preferred direction in motion detection is less

desirable than that of Fig 24 since motion in the null direction does not produce a true null. This is probably not a serious drawback. One method of improving the null might be to place another processing stage in series.

The best motion detection scheme in terms of saving precious retinal space would be to have the retinal lateral inhibition network function simply in a spatial role (sharpening, contrast enhancing, and dynamic range increasing). The gaglion cell outputs would then fan out to subsequent central lateral inhibition networks where null versus preferred direction as well as high pass or low pass characteristics would be developed.

The lengthy discussion of input stimulus representation for lateral inhibition networks would apply centrally, but would probably not be necessary retinally, where predominantly slow potentials occur. With slow potentials, the problems of regularity and phase would not occur. But if ganglion cell outputs were fed to central lateral inhibition networks, such considerations would apply.

Although handicapped by the basic difference in form of input stimulus, the LINHIB program did a good job in exploring the effects of line sharpening, inhibition and disinhibition, and Mach band formation. It must be emphasized, however, that LINHIB models a central lateral inhibition network, whereas most of these spatial phenomena probably occur retinally. The modeling of spatial phenomena should, therefore, be taken less seriously than the modeling of temporal phenomena.

The performance of LINHIB in suppressing low spatial frequencies was not as dramatic as expected until performance without inhibition was explored. Then, it became obvious that much of the low spatial frequency suppression

was being masked by a change in the basic EIPSP neuron's output/input ratio as input density increases. To belabor the point, the same change in output/input ratio would in all probability not occur with slow potential interaction, and the low spatial frequency suppression would be more marked. This represents additional evidence that low spatial frequencies are suppressed retinally.

VI. RECOMMENDATIONS FOR FUTURE MODELING WORK

Many physiological systems are ripe for computer modeling. One example is the muscle stretch receptor unit. Comments here, however, will be confined to expanding and refining the modeling of visual neurophysiology, and the neuron in general.

At present, all modeling assumes that information passes from neuron to neuron via the chemical synapse, and that all information is in the form of action potentials or spikes. There is a growing body of evidence that other forms of inter-neuronal communications exist. It is a long established fact that sensory receptors do not generate spikes, but rather maintain a greater or lesser membrane potential in response to external stimuli. Similarly, horizontal and bipolar cells in the retina do not generally exhibit spike activity, but communicate with other neurons by some other means. Recent evidence indicates that the varying membrane potential in these cases modulates the release of neurotransmitter at chemical synapses [Ref 15]. Although this neurotransmitter has not yet been specified for retinal neurons and receptors, the synaptic vessicles can clearly be seen in electron micrographs. It appears to be a valid general principle that spikes are not necessary for inter-neuronal communication, but rather that their purpose is to allow a summed signal at the axon hillock to be communicated intra-neuronally to the presynaptic bulbs via a lengthy axon. Electrotonic spread would be inadequate because of the losses which would occur over the length of the axon. In order to be as factual as possible, then, the modeler should undertake this sort of inter-neuronal

communicatien.

The fact that loss is inherent in electrotonic spread of the PSR raises an interesting issue. Namely, how much membrane potential change at the synapse is necessary to cause modulation of neurotransmitter release? With spike action potentials, arrival is accompanied by the release of one "unit" of neurotransmitter. With slow potentials, however, the length of the axon-like process would affect the change in potential at the synapse, and therefore would also affect the degree of modulation of neurotransmitter release. It is not entirely reasonable that process length should be so important, but it would serve as a mechanism whereby the strength of inhibition would decrease with distance from the neuron initiating the inhibition. Perhaps there is some threshold for membrane potential change, and so long as the change were greater than threshold, a "unit" of modulation would occur. It is doubtful that this point could be clarified by the type of modeling described in this thesis.

In addition to non-spike inter-neuronal communication at chemical synapses, it has been maintained for many years that electrical synapses exist. The major evidence seems to be twofold. First, specialized regions at which membranes come into ultra close proximity (on the order of 20 angstroms) have been observed, and are believed to be electrical synapses. Second, microelectrode studies in many instances have produced neuron pairs whose PSP's vary nearly synchronously, but not identically, [Ref 14] in response to induced membrane currents in only one of the neurons. The principle drawback to quick acceptance of this scheme is that the mechanism of coupling has not been elucidated. If a current at the electrical synapse is responsible for the change in membrane potential of the postsynaptic neuron, then the unquestioned existence of membrane capacitance

(which would require an integration time in order for synaptic current to produce a postsynaptic voltage change) would make simultaneous variation of membrane potentials impossible. If current at such a specialized site causes some postsynaptic membrane permeability change which would then spread, causing a rapid change in PSP, this would be a more plausable explanation. No such mechanism has been found, however. Because electrotonic coupling seems to be here to stay, the modeler should include it in his repertoire.

One improvement in the present way the PSP is computed might be in order. Currently, a large number of closely spaced I-inputs will drive the PSP negative until the largest negative integer capable of being represented by one computer word is reached. In a real neuron, however, there is a limit to the amount of hyperpolarization which can occur. It is the ion concentrations, both inside and outside the neuron, as well as the membrane's permeability for each ion which, when plugged into the Nernst equation [Ref 9], give membrane potential. Excitatory as well as inhibitory inputs alter membrane potential by altering the membrane's permeability for sodium and/or potassium ions. Thus, there is a limit to the amount of hyperpolarization which can occur; this limit should be incorporated into the models.

There is one improvement which could be made in the fatigue/facilitation program (PSPFAT). At present, the running average is calculated for each word of the 1K processing block. That number is then used to adjust the magnitude of the PSR for all inputs since the end of the last refractory period. Thus, the shape of the PSR for a given input would vary from the accepted shape because of its being weighted differently at different processing words. A more realistic method, but one which involves more processing, would be to assign a weight to the PSR of a

given input at the time the input occurs. Then, whenever that particular PSR were summed to form a PSP word, the original weight would be used. This could be handled by using the high bytes of the words of the age factor stack as the weight. This method would be more consistent with the mechanism of fatigue in which the amount of neurotransmitter released changes.

As mentioned in the section on motion detection, the null versus preferred detector using LINHIB differs from that propcsed by Ref 12 and pictured in Fig 24. This neural circuit would be very worthwhile to model.

Discussed in the section on oscillatory PSP's was the problem of phase in a speed band pass gate. The thought that perhaps the oscillator neuron is strobed by a moving target of any speed such that the proper phase is produced in the oscillatory PSP is an intriguing one well worth an attempt at modeling.

The mcving target models using LINHIB all have one difficulty which should eventually be corrected. For a mcving target, the models work satisfactorily. But consider the response to a bright stimulus which simultaneously illuminates all eight neurons. All would have outputs, and the speed gate target neuron would have a false sense of a moving target at the pass speed. The solution to this is to introduce fatigue into each of the eight neurons. In this way, significant firing would occur in all eight only initially, and the target neuron would have output only at first.

Another improvement to LINHIB would involve the directionality of the ramp delay. At present, either constant delay in both directions or ramp delay in both directions is selectable. If one could select constant delay

in one direction and ramp delay in the other, then a neuron with null and preferred directions which could have high speed pass properties in the preferred direction could be modeled. Similarly, if different PSR durations could be specified for different directions in LINHIB, then a network which could have a null direction, and detect only slow targets in the preferred direction could be modeled.

With only eight channels available in LINHIB, the investigation of spatial phenomena is severely restricted. Any meaningful evaluation of the spatial frequency response of such a network would require far more than eight channels of resolution. If 100 channels were available, one could provide inputs which had various spatial frequency characteristics (such as a sinusoidal or square wave grating), and derive the outputs. The spectra for input and output could then be found by Fourier transform of a block of 100 words where each word represents the count in a channel, thus providing a transfer function in the frequency domain. The parameters to be varied would involve the distance of inhibitory spread and the spatial shape of the fall-off. Using the general philosophy of LINHIB, but requiring enlargement, rearrangement, and reorganization, one could expand the number of channels to a very large number. This would involve shortening the block length, perhaps, but would also require the technique of processing all blocks for several words, storing results on disk, processing the next several words for all blocks, and so on until complete. Plotting would then require recall of blocks from the disk. This modeling project would be an undertaking of major proportions.

Neural circuits which recognize static patterns exist and should be fairly easy to model. Examples of static patterns which have been observed to produce strong outputs from cortical target cells are borders between light and

dark regions and light or dark slits or narrow bars on contrasting backgrounds. Pattern orientation is of extreme importance in these examples [Ref 10].

The importance of pattern recognition to living organisms cannot be overstated. It is amazing that a person can recognize a dinner plate even though it is tilted such that it appears elliptical. Even more amazing is the fact that an oval serving platter also appears elliptical when tilted, but this would seldom be confused with the elliptical shape of the dinner plate. It is true that this sort of recognition would require far more complicated neural circuitry than recognition of a slit or border. Nonetheless, the principles are probably the same. The target neurons of cortical area 17 (the primary visual area) identify small simple patterns. The columnar organization of area 17 allows cortical area 18 (the secondary visual area) to piece together long lines, bars, borders, and shapes from short patterns. Cortical area 19 (the visual association area) performs the tasks of putting long lines and shapes together into an overall perception, and of bringing the individual's past visual experiences into comparison with the current scene. This is a giant step representing a massive amount of processing, but in all likelihood, many repetitions of a few simple circuits are responsible for it.

The modeling of pattern recognition circuits is a large area, and a very interesting one, as it relates so closely to one's most valuable sense. Creativity in devising and modeling neural circuits capable of recognizing patterns could aid in understanding the neural circuits which perform pattern recognition in real visual systems. The modeling of pattern recognition circuits is certainly one of the most worthwhile areas for future modeling.

LIST OF REFERENCES

1. Barlow, H. B., Hill, R. M., and Levick, W. R., "Retinal Ganglion Cells Responding Selectively to Direction and Speed of Image Motion in the Rabbit," Journal of Physiology, v. 173, p. 377-407, 1964.

2. Bullock, T. H., "Neuron Doctrine and Electrophysiology," Science, v. 129, p. 997-1002, 17 April 1959.

3. Campbell, F. W., and Maffei, L., "Contrast and Spatial Frequency," Scientific American, v. 231, p. 106-114, November 1974.

4. Carew, T. J., and Kandel, E. R., "Two Functional Effects of Decreased Conductance EPSP's: Synaptic Augmentation and Increased Electrotonic Coupling," Science, v. 192, p. 150-153, 9 April 1976.

5. Carney, J. M., Computer Modeling of Basic Neural Circuits, MSEE Thesis, Naval Postgraduate School, Monterey, 1976.

6. Cornsweet, T. N., Visual Perception, Academic Press, 1970.

7. Eccles, J. C., The Physiology of Synapses, Academic Press, 1964.

8. Glickstein, M., and Gibson, A. R., "Visual Cells in the Pons of the Brain," Scientific American, v. 235, p. 90-98, November 1976.

9. Guyton, A. C., Textbook of Medical Physiology ,

Saunders, 1971.

10. Hubel, D. H., "The Visual Cortex of the Brain," Scientific American, v. 209, p. 54-62, November 1963.

11. McClane, J. L., Biofeedback Related to Enhancement of Preferred Frequencies in the Electroencephalogram, MSEE Thesis, Naval Postgraduate School, Monterey, 1976.

12. Michael, C. R., "Retinal Processing of Visual Images," Scientific American, v. 220, p. 105-114, May 1969.

13. Ratliff, F., "Contour and Contrast," Scientific American, v. 226, p. 91-101, June 1972.

14. Spira, M. E., Spray, D. C., and Bennett, M. V. L., "Electrotonic Coupling: Effective Sign Reversal by Inhibitory Neurons ," Science, v. 194, p. 1065-1067, 1o December 1976.

15. Tomita, T., and others, "Electrical Response of Retinal Ganglion Cells as a Sign of Transport," Experimental Eye Research, v. 16, p. 327-341, 1973.

16. Weight, F. F., and Erulkar, S. D., "Modulation of Synaptic Transmitter Release by Repetitive Postsynaptic Action Potentials," Science, v. 193, p. 1023-1025, 10 September 1976.

17. Werblin, F. S., "The Control of Sensitivity in the Retina," Scientific American, v. 228, p. 71-79, January 1973.

18. Williams, P. L., and Warwick, R., Functional Neuroanatomy of Man , Saunders, 1975.

19. Young, R. W., "Visual Cells," Scientific American, v. 223, p. 79-92, October 1970.

INITIAL DISTRIBUTION LIST

		No. Copies
1.	Defense Documentation Center Cameron Station Alexandria, Virginia 22314	2
2.	Library, Code 0212 Naval Postgraduate School Monterey, California 93940	2
3.	Department Chairman, Code 62 Department of Electrical Engineering Naval Postgraduate School Monterey, California 93940	1
4.	Professor George Marmont, Code 62 Department of Electrical Engineering Naval Postgraduate School Monterey, California 93940	10
5.	LCDR Seaborn M. McCrory, III, USN (Student) 19110 Creekside Place Salinas, California 93901	1
6.	Office of Naval Research Attn. CAPT John C. Bajus, USN, Code 101 800 N. Quincy Street Arlington, Virginia 22217	1
7.	Commander Naval Electronics Laboratory Center Attn. LCDR S.E. Dollar, USN, Code 1300 San Diego, California 92152	1

8. Naval Electronics Systems Command 1
Attn. CAPT James Wheeler, USN, Code 03
Navy Department
Washington, D.C. 20632

9. Naval Electronics Systems Command 1
Attn. J. S. Lawson, Jr., (NAVELEX 00B)
Navy Department
Washington, D.C. 20632

Thesis 169753
M18255 McCrory
c.1 Computer modeling
the neurophysiology
of vision.